Introduction

This chapter is not concerned with cardiothoracic pathology and management that are thoroughly discussed in medical books, nor is intended to supply a course on the detailed surgical indications and techniques which are, of course, beyond the scope of medical students. Our aim was to highlight some of the important subjects from the cardiothoracic surgeon point of view and hence, sharpen the student's awareness on when, and generally how, those illnesses can be dealt with surgically.

While management of chest trauma, empyema thoracis and mediastinal diseases were dealt with in some details, only the surgical indications and their results were discussed for patients with bronchogenic carcinoma. The basic principles of cardiopulmonary bypass were given to simply know: how it works? In fact, cardiopulmonary bypass is becoming, in itself, a large field of research. Coronary bypass grafting is actually the commonest cardiac operation performed worldwide and hence its indications, techniques and results were highlighted. In aortic aneurysm and dissection section, emphases were made on indications and appropriate timing of surgery. We did not miss the opportunity to briefly discus blood conservations techniques and the general guidelines for cerebral as well as other organ protection during surgery. In their broader lines, special techniques of aneurysm surgery were supplemented for the interested reader. On the other hand, the still endemic rheumatic heart disease obliged us to discus the indications as well as the inherent complications of cardiac valve replacement. Most importantly, was to give the necessary precautions undertaken when those many patients become subjected to non-cardiac surgery or instrumentation. In the congenital heart section we discussed in short some rewarding surgeries that offers complete cure of a simple pathology, not to mention the difficulties encountered when operating upon the young enfant with complicated cardiac morphology. Being a habit, CPR was included in this course; even if we believe that other specialties are equally interested. We advise the reader to attend the practical course held at our department for consolidation and refinement of the given knowledge and to review the selected web sites for further readings.

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Management of Chest Trauma

Although chest injuries are common during war time and are responsible for about 25% of battle field casualties, yet they are actually becoming a large part of our civil practice for the progressively increasing road accidents. The cause is usually severe and hence, they are seldom present alone and are often associated with abdominal and/or head injuries.

A blunt chest trauma may be directly caused by a blunt object or a crush injury as well as indirectly by explosions through the generated strong concussion waves. Despite the absence of open wounds, the injury is usually severe and can affect all thoracic contents.

1- The bony cage can be broken in a single or multiple sites and its seriousness can vary from simple or multiple ribs fracture to the serious stove-in chest, flail chest and spine fracture.

2- Air escaping from the injured lung tissue may appear in the mediastinum (pneumomediastinum) or under the skin (surgical emphysema); but more often in the pleural space compressing the involved lung (pneumothorax) and causing respiratory distress. The emergency situation in which air compresses both: the involved lung and the mediastinum and then encroach upon the contra-lateral lung is the life threatening condition known as tension pneumothorax.

3- The chest wall muscles, lungs, diaphragm and other thoracic soft tissues may suffer from contusions and lacerations, which can either be treated conservatively or may be severe enough to necessitate chest wall or diaphragmatic repair as well as varieties of lung resection.

4- The severity of injury may even lead to rupture of the deeply seated and protected chest wall arteries and veins. Rupture can affect a major vessel like the large thoracic aorta with a resulting exsanguinating hemorrhage and profound shock to a small coronary artery that can sometimes lead to myocardial infarction.

The other variety of chest trauma is the **open type** resulting from a stab wound, gun-shot, etc… All the previously mentioned clinical forms (rib fracture, pneumothorax, lung contusion, myocardial infarction, etc…) can equally result from an open type injury. In fact, both closed (blunt, crush injury, blast injury,…) and open types can coexist and the management will depend upon the general condition of the patient, the clinical type and severity of the resulting injury, the associated other organ diseases or injuries as

well as the hospital facility, equipment and experience of working team. The general guidelines include:

1- Airway control: Trauma may seriously interfere with the patient respiratory and coughing ability due to pain and chest wall deformity, with accumulation of tracheo-bronchial secretions and blood which further aggravate the respiratory limitations in a vicious circle manner. Soon hypoxia can settle down and respiration becomes shallow and rapid with cyanosis, tachycardia and hypotension. Hence, the first line of management is to keep a patent respiratory airway. This involves cleaning of airway passages through rapid nasal and oral toilet, suction of any blood clots or debris and supplying oxygen through nasal oxygen mask. Bronchospic lavage may sometimes be necessary and, if need to be repeated, tracheosotomy has to be considered.

In a hospital with a trauma facility center, the patient should be connected to a pulse oxymetry monitor to continuously verify his blood oxygen saturation and arterial blood gazes sample should be drawn to verify his Po2, Pco2, Ph and base deficit. The presence of hypoxia (Po2 less than 60 mmHg) or hypercapnea (Pco2 more than 50 mm Hg) points to respiratory failure and, unless the cause is promptly found and treated, endotracheal intubation and mechanical ventilation are indicated. If the patient is also in shock, intubation becomes a must and the golden rule is to intubate the trauma patient in case of doubt.

2- In the initial survey, the chest wall is inspected for open or sucking wounds, for crepitations denoting air escape under the skin, for bullet inlet or exit in case of fire arm injury and for the shift of the normal position of the mediastinum (central trachea and apex beat in the 5^{th} intercostal space) to the contra-lateral side for the presence of pneumothorax or massive hemothorax. The type of chest wall deformity is noted: paradoxical movement in case of flail chest and depressions in case of stove in chest. The back is also carefully examined for associated fractures.

3- Obtain a chest x-ray that should be taken classically in the poster-anterior view and with the patient in the erect or sitting position; where a hemothorax is shown to obliterate the costophrenic angle. Such diagnostic obliteration can be lost -and hemothorax can be consequently passed by- if the patient was lying flat on his back during x-ray exposure.

4- Prompt drainage of pleural blood or air to decompress the lungs. Although simple needle drainage of hemothorax is done first, yet this is usually followed by the insertion of a wide bore intercostal tube in a dependent area to insure full drainage

(usually the 5th intercostal space in mid-axillary position). On the other hand, and In case of pneumothorax, a smaller sized intercostal tube has to be directed up towards the lung apex to drain the (light weight) leaking air. In case of hemopneumothorax, the intercostal tube is directed to drain blood primarily.

5- Pain relief with small doses of analgesics or intercostal nerve block.

6- Venous access lines to transfer blood and fluids and to measure the central venous pressure. In case of a severe trauma or shock, a transurethral catheter is inserted, urine is collected and measured hourly and a fluid balance chart is created.

7- Arrange for ECG as well as continuous ECG monitoring and in severe cases, the patient's arterial blood pressure has to be continuously and invasively monitored too. If myocardial injury is suspected, cardiac enzymes (CPK-MB, LDH and troponin) should be measured for possible myocardial ischemia.

8- In severe cases, consult other specialists for associated neurological, orthopedic, cardiac and respiratory injuries and never miss early anesthesiologist consultation for possible surgical interference. In this setting, arrange for ample blood bank facilities and reserve an ICU bed. Further investigation and management will then depend upon the clinical presentation of the injury (vide-infra).

A- The Thoracic Cage

Rib fracture: The arch design of the ribs allows for some flexing, more so in children than adults, which can absorb small amounts of blunt kinetic energy. Crush or rollover injuries or significant deceleration injury commonly breaks a rib in 1 position, but only a significant impact breaks a rib in 2 or more positions. Localized tenderness and crepitus are often elicited and the plain radiograph is the best means for evaluation and exclusion of associated injuries. Isolated rib fracture is not of great clinical importance other than the considerable pain it can cause the patient and hence, local support and analgesics are all what is required and nerve block is rarely indicated for persistent pain.

Specific rib fractures of concern are those of the 1st and 2nd ribs, of the clavicle and those causing flail chests. The importance of the 1st and 2nd ribs as well as of the clavicle is that they are difficult to break. Thus although the fractures themselves are of little consequence (unless they impinge on or damage the vessels and nerves of the thoracic inlet), they indicate that a considerable force was applied and should raise the suspicion of more serious injuries to the aorta and other structures.

Multiple rib fractures however, can disturb chest wall stability and the presence of severe pain can lead to paradoxical movement, inappropriate ventilation, hypoxia, Co2 retention and retained secretions. Careful respiratory care and analgesics are usually sufficient however; tracheotomy is sometimes indicated for correction of paradoxical respiration by abolishing the dead space and allowance for a ready route for tracheo- bronchial aspiration and artificial ventilation.

Flail chest is likely the most common serious injury to the thorax. It is traditionally described as the paradoxical movement of a segment of chest wall caused by fractures of 3 or more ribs anteriorly and posteriorly within each rib due to a significant blunt trauma (figure 1). In fact, flail chest requires significant blunt force trauma to the torso to fracture the ribs in multiple areas, such as motor vehicle accidents, falls, and assaults in younger, healthy patients. On the other hand, flail chest may occur with lesser trauma in persons with underlying pathology, including osteoporosis, total sternectomy, and multiple myeloma.

At first, the actual paradoxical movement of the flail segment is limited by the surrounding muscolo-skeletal component but as the lungs stiffen due to atelectasis and accumulation of secretions, inward motion of the ribs will become more pronounced with inspiration. As the patient breathes in, the negative pressure “sucks in” the unstable segment. This is usually not harmful until increased ventilatory pressures are required, as with partial airway obstruction or underlying pulmonary contusion. As the patient’s pulmonary condition worsens, the paradoxical rib motion becomes more severe, making respiration inefficient. The unconscious patient, who does not use the chest wall muscles to “splint” the injured area, will have a more pronounced flail effect.

For long time it was thought that respiratory insufficiency in flail chest was due to the “chest wall defect”, however it is the severity of the underlying lung injury and not the flail segment that causes a problem and respiratory insufficiency in flail chest is much more likely to be a result of the underlying severity of pulmonary contusion and ventilation perfusion mismatch. Thus, the adept surgeon usually looks past the structural deformity and determines the physiologic compromise caused by the pain of the rib fractures, the tidal volume changes, and the underlying pulmonary and cardiac injury.

Chest x-ray occasionally demonstrates the fractured ribs but may not show all fracture sites, and underlying pulmonary contusion may be initially masked by hypovolemia. Because many of these patients sustain concomitant internal thoracic

injury, thoracic CT scanning images may be available for reasons other than rib fracture identification. Arterial blood gas measurements show the severity of the hypoventilation created by both the pulmonary contusion and the pain of the rib fractures, and are helpful at baseline to assess the need for mechanical ventilation and to follow the patient during management.

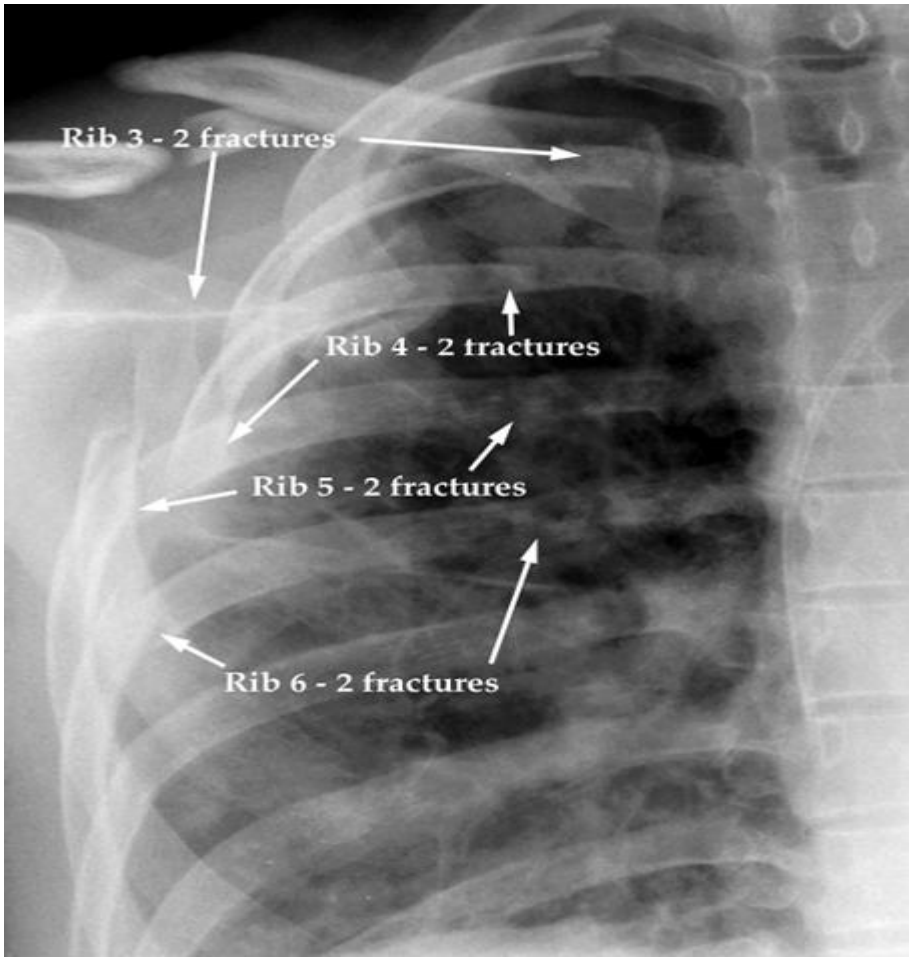

Figure 1: Flail chest. Chest X-ray in PA view showing multiple ribs fracture both: anteriorly and posteriorly.

Many patients fared better with adequate pain control and pulmonary toilet (including medical management of their pulmonary injury), and mechanical ventilation is reserved for patients with persistent respiratory insufficiency or failure after adequate pain control or when complications related to excessive narcotic use occur.

Surgical stabilization of the rib ends by stainless steel wire or nails is an option that is rarely necessary. As previously noted in traumatic causes however, the severity of respiratory failure is less a result of either the paradoxical motion of the chest wall (tidal volume abnormalities) or chest wall instability. Accordingly, surgical stabilization is not routinely indicated and is most commonly performed in patients requiring a thoracotomy for other reasons or in cases of gross chest wall deformity.

Stove-in chest an extensive localized crushing force may produce multiple rib fractures with inwards indentation of the chest wall. Although some paradoxical chest movement may be present but, as in flail chest, its mainly the disturbed ventilation of the lung underlying the affected hemi thorax that opens the vicious circle for hypo perfusion, retention of secretions and hypoxia. The lines of management are as those applied for fail chest and, equally, surgical retraction of the affected rib segments is rarely indicated.

Chest wounds: An open wound allows air to be sucked into the chest with inspiration. If large enough, it may interfere with air motion in the lungs by decreasing the amount of negative pressure that can be generated during inspiration. Small wounds can form one-way valves that allow air to be sucked and trapped inside the pleural cavity, leading to tension pneumothorax. Clinically, an open chest wound will usually exhibit some unusual motion during respiration, such as retraction, shaking, or burping.

The diagnosis is made on clinical inspection of the wound. X-ray will show a pneumothorax, but action must be taken before the x-ray. The wound should be covered with an occlusive dressing such as Vaseline gauze and the dressing should be taped on three sides, so it can act as a one-way valve allowing air to exit the chest with expiration, but preventing sucking-in during inspiration. An intercostal chest tube connected to a closed under water seal system should be placed at a second site so as to evacuate the collecting air and/or blood. The rule is never to use the wound itself for the tube -even if its size is perfect- because of contamination.

The intercostal tube should be monitored for the amount of blood loss and an increasing rate is an indication for surgical exploration. Although these simple wounds seldom need surgical interference, yet complicated injuries with large missiles commonly dictates an exploratory thoracotomy (sometimes even extended to a thoracoabdominal incision), for the repair associated considerable chest wall damage, lung contusions, and involvement of mediastinal structures, diaphragm and even abdominal viscera.

Thoracic spine fractures account for 25-30% of all spine fractures and usually occur at the thoracolumbar junction, roughly T9-T11. This is where the articulating facets change from a “cervical” to a “lumbar” pattern with resulting stiffening of the spine. Fractures of this region are particularly serious because the spinal cord occupies a greater percentage of the cross sectional area of the spinal canal than it does in

either the cervical or lumbar regions. This makes it particularly susceptible to injury from displaced bone fragments or malaligned vertebral bodies. Additionally, the blood supply to this part of the cord is rather tenuous and an apparently small injury can disrupt the blood supply to a large portion of the cord.

B- The Pleural Space

Hemothorax: Free blood in the chest usually comes from cut or torn pulmonary and thoracic vessels and is a common association with chest trauma that is seen in as 25-50% of blunt trauma and up to 75% of penetrating trauma victims. The fluid is usually clinically suspected by the presence of decreased breath sounds, dullness to percussion, mediastinal shift on physical exam and possible hypotension. Hemothorax is distinguished from pneumothorax by dullness on percussion. Chest x-ray may confirm the diagnosis (figure 2), but as previously mentioned, an upright or decubitus film is often necessary. Up to a liter of blood may be present, and not seen, on a standard portable supine chest x-ray. In the x-ray, hemothorax appears as a whitish opacity with its tail rising towards the axilla. For an adult, just obliteration of the costophrenic angle indicates that as much as 500 ml of blood are present in the pleural cavity. It is always advisable to remove the blood of a moderate-sized hemothorax, even if the blood accumulation is not enough to interfere with respiration; as thrombolytic substances are released by the old blood and bleeding often continues.

Figure 2: Right -sided hemothorax in PA (left) and right lateral (right) views.

Diagnostic needle aspiration is done preferentially at 2 sites: the 5^{th} or 6^{th} intercostal space mid-axillary line and the infra-scapular space and care is taken not to introduce air during the procedure. The presence of a free flow of blood is an indication

for the insertion of an intercostal tube for total drainage of blood and is usually performed through the 5th or 6th intercostal space mid-axillary line too. The tube should be large (36-40), dependent, aimed posteriorly and is connected to an under-water seal to prevent the introduction of atmospheric air inside the chest cavity. In addition to achieving complete drainage of old blood which is a source of thrombolytic factors that invites more bleeding; placement of a chest tube also serves to tamponade bleeding by bringing the lung surface up against the chest wall. Only 10-15% of patients with traumatic hemothorax will need a thoracotomy because of massive initial hemothorax (>1000ml) or because of continued heavy bleeding.

Pneumothorax: The importance of a pneumothorax is determined not by its size, but by its physiologic effect. An otherwise healthy person can tolerate a unilateral totally collapsed lung without problem whereas a person with an underlying lung disease may have extreme problems with a very small pneumothorax. A trauma patient, however, is a special case because even a small pneumothorax can quickly become life threatening when a patient is placed on mechanical ventilation or given general anesthesia. Thus, pneumothorax must be looked for meticulously and the clinical service alerted, even if the patient is not symptomatic.

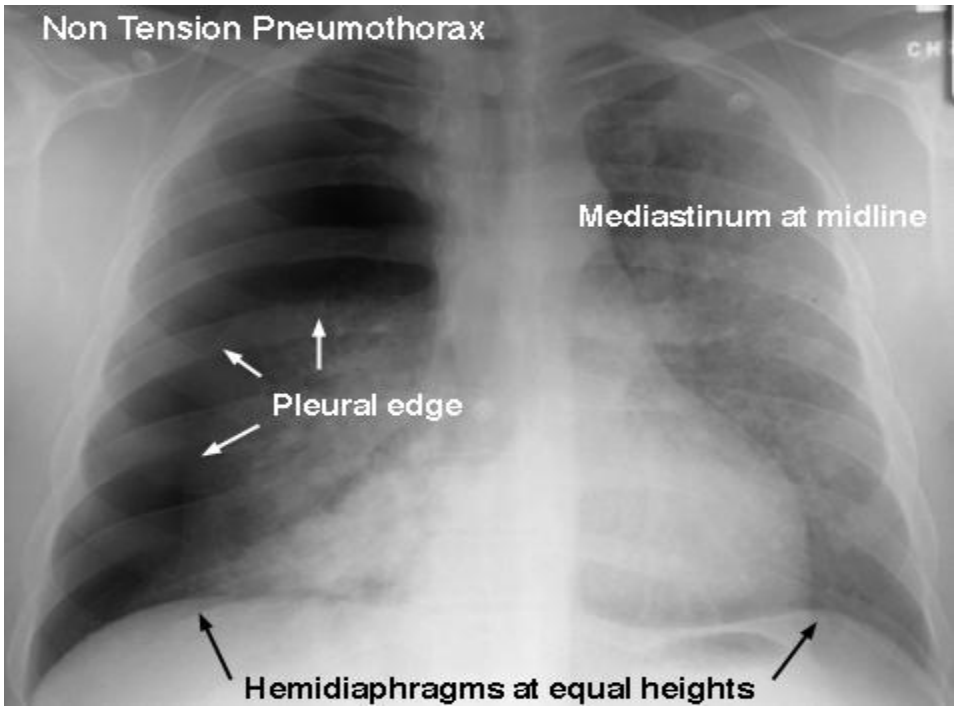

Figure 3: Chest x-ray (PA view) showing non-tension pneumothorax on the right side

Generally, 20% of penetrating trauma patients and up to 40% of blunt trauma patients will have pneumothorax. Free air in the pleural space will travel to the least dependent portion of the chest. In an upright person, this will be at the apex of the lung and is usually easily identified when compared to the adjacent lung (figure 3). Although an apical pneumothorax is generally easy to see, many trauma patients cannot be placed in an upright position and are studied supine. Thus the least dependent part of the chest is not the apex, but rather the base of the chest at the level of the diaphragms. Signs of free air in this location include the “deep sulcus” sign where the costophrenic sulcus is significantly lower than on the contra lateral side, hyperlucency of the lung base due to the free air, and unusually clear definition of the diaphragm and cardiac apex. The whitish edge of the collapsed lung will appear away from the chest wall and the lung itself is folded upon itself and is shifted towards the mediastinum. In tension pneumothorax, the mediastinum itself is further pushed to the other side (shift of the normally central dark air column present in the trachea) encroaching upon the contra-lateral lung (figure 4). Although CT is much more sensitive for pneumothorax than plain radiography, it is rarely used solely to look for free air.

Accumulation of air within the pleural space may compromise respiration by interfering with the expansion of the lung. Respiratory distress usually is not seen until the pneumothorax exceeds 40% of one lung’s volume, unless the patient has pre-existing lung disease or parenchymal lung injury. Clinically, breath sounds will be decreased on the affected side. Pleuritic pain may not develop for hours. If the pneumothorax is large, hyperresonance to percussion may be present. In tension pneumothorax, pressure increases within the pleural space, venous return to the chest slows, and shock develops. The triad of respiratory distress, shock, unilateral absence of breath sounds that is hyper resonant to percussion is diagnostic to tension pneumothorax.

A very small pneumothorax with the lung border only 1-2 cm away from the chest wall on the x-ray and in an otherwise healthy individual; can be observed without treatment if it remains stable on subsequent x-rays (6-8 hours later). Otherwise, evacuate the pneumothorax by chest tube insertion and under water-seal drainage of air. On the other hand, and even before acquiring the chest x-ray in a patient showing the triad of tension pneumothorax, a needle inserted in the second intercostal space anteriorly reveals the hiss of escaping air and establishes the diagnosis. Nevertheless,

Tension pneumothorax is a surgical emergency that requires immediate drainage through chest tube insertion as a life saving procedure.

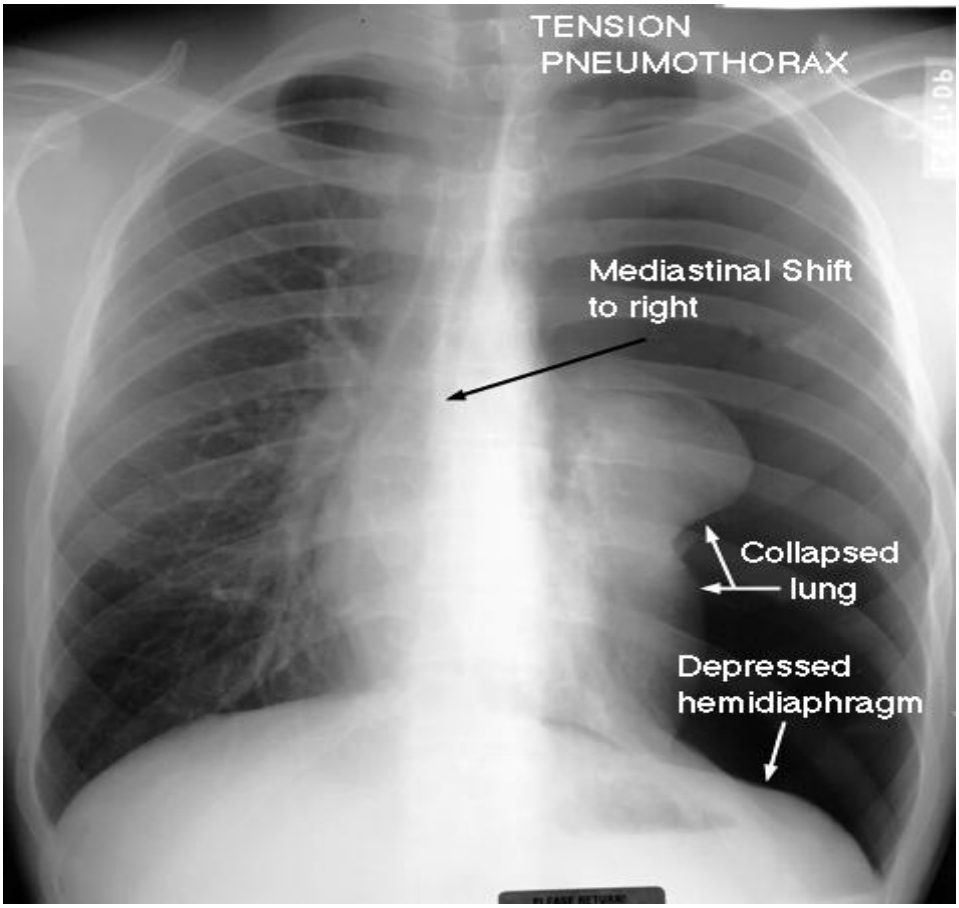

Figure 4: Chest x-ray (PA view) showing tension pneumothorax

C- The Thoracic contents

All thoracic contents are liable for injury especially the lungs, heart, blood vessels, esophagus, thoracic duct and diaphragm.

Pulmonary Contusion and lacerations: Bruising of the lung results from passage of a shock wave through the tissue. Microscopic disruption occurs at any air-tissue interface — of which the lungs have plenty. Injuries involving high velocity rather than slow crushing are more likely to cause pulmonary contusion. Clinically, Rales will often be heard. The chest x-ray shows opacity in the peripheral lung near to the injured chest wall. The chest x-ray may lag 12-24 hours behind the clinical extent of the contusion. Blood gases will tend to worsen for two or three days as edema increases in the lung. Stiffness of the lung causes dyspnea and elevated respiratory rate. The diagnosis is made when parenchymal infiltrate is seen adjacent to injured chest wall. It is worth noting that pulmonary contusion may exist, despite a normal x-ray.

Mild cases are treated with oxygen and observation. If respiratory distress is present, intubation and mechanical ventilation are beneficial while the lung recovers. One has to be aggressive in treating patients who have pulmonary contusion combined with severe abdominal injuries or COPD. Pulmonary lacerations can heal spontaneously, but severe forms will cause persistent lung collapse and air leakage and need exploratory thoracotomy that usually involves repair or section of the affected parts. This is also the case where bronchial fracture, most commonly occurs at the junction of the relatively fixed mediastinal bronchus and the relatively mobile hilar bronchus with total lung atelectasis and mediastinal emphysema. In this setting however, early surgical repair of the bronchus is a must before the settling of an irreversible infection and fibrosis.

Cardiac injury can be seen without severe anterior chest injury. Myocardial contusion is a physical bruising of the cardiac muscle. The patient is prone to all the complications possible with acute myocardial infarction. Virtually all patients with chest pain following blunt trauma should have an ECG which may show an ST elevation. If no other injuries requiring hospitalization are present, the decision to hospitalize the chest-injury patient to rule out myocardial contusion must be based on the clinical picture, for example, the presence of substernal pain or a bent steering column. These patients should be hospitalized for cardiac monitoring and serial enzyme determinations.

Cardiac Tamponade: Pericardial blood is usually due to penetrating injuries of the heart. In those cases that are not rapidly fatal, the pericardium may seal and the increasing pressure in the pericardial space prevents further bleeding from the cardiac wound. This pressure prevents the heart from filling completely, and obstructive shock can occur. The classic triad of distended neck veins, low blood pressure, and muffled heart tones is present in less than a third of patients. Neck veins may not be distended if hypovolemia is present, and muffled heart tones are often not present. Pulsus paradoxus (a 10-15 mm drop in systolic blood pressure with inspiration) may also be present.

Diagnosis is further suspected when the patient does not respond to fluid resuscitation (and in which tension pneumothorax is ruled out). X-rays are usually normal and the diagnosis is established by echocardiography. Treatment is achieved by exploration and surgical repair of the source of bleeding.

Aortic Rupture is the traumatic laceration of all three layers of the aortic wall, intima, media, and adventitia. This is in distinction of aortic tear which is laceration of

the intima and media, but not the adventitia. In a rupture there is free communication between the lumen of the aorta and the mediastinum; in a tear the blood is contained by the adventitia. Clearly, given the fine nature of the adventitia an aortic tear can become a rupture easily.

About 90 percent of patients with aortic rupture die within minutes, and of those who arrive at the hospital alive, another 90% will die. Most ruptures are due to auto accidents, with the site of rupture just beyond the left subclavian artery near the ligamentum arteriosum. The mechanism of injury is sudden motion of the heart and great vessels within the thorax, rather than direct crushing. Many patients will therefore have little external evidence of serious chest trauma.

Physical exam findings are rarely helpful. Weak leg pulses with hypertension in the arms, or a new murmur may suggest the diagnosis. Aortic rupture is more likely in patients with 1st or 2nd rib fractures. Chest x-ray may show deviation of the NG tube 1-2 cm to the right or blurring of the aortic knob. A widened upper mediastinum, deviation of the trachea to the right, a “pleural cap,” or loss of the clear space between the aorta and pulmonary artery may also suggest the diagnosis. The critical condition of the patient rarely permits Aortography and both: Echocardiography and chest CAT with contrast are sufficient for the diagnosis. Treatment consists of rapid fluid resuscitation and prompt surgical repair in a specialized centre with cardiopulmonary bypass facility.

Diaphragmatic rupture and Traumatic diaphragmatic hernia: The diaphragm may be torn directly by a missile as a part of a thoracoabdominal wound or ruptured by a severe blunt lower thoracic or abdominal crush injury. In the latter case, the weak dome of the diaphragm is the preferred site of rupture. Diaphragmatic rupture likely occurs as a result of a sudden and massive increase in intra-abdominal pressure. If the diaphragmatic tear is large enough, the relatively negative pleural pressure during spontaneous ventilation encourages herniation of abdominal organs into the chest compressing the adjacent lung leading to respiratory embarrassment. Diaphragmatic injuries are more frequent on the left side as the liver affords some protection to the right hemidiaphragm. Physical examination is not very helpful in making the diagnosis of diaphragmatic injury in the patient with multiple injuries.

Chest radiography demonstrating elevation of the left hemidiaphragm with an arch-like shadow suggesting an abnormally high diaphragm, bowel gas patterns in the left hemithorax and shift of the heart and mediastinal structures to the opposite side of

the defect suggest a diaphragmatic injury. Diagnosis can be confirmed by passing a nasogastric tube as the tip of the tube may be seen in an intra-thoracic stomach.

Tracheal intubation and positive pressure ventilation will alleviate respiratory distress and protect the airway. The hernia should be surgically repaired, viscera pushed down to the abdominal cavity, diaphragm reconstructed with strong interrupted sutures buttressed with Teflon pledgets and the compressed lung is reinflated.

Esophageal Injury related to blunt external trauma is usually quite rare. Injury may be the result of rapid compression of the abdomen, which may raise pressures in the stomach to such a degree that an intraluminal tear of the esophagus results. Another mechanism is accelerated insufflations of pressured air into the esophagus in an attempt to ventilate the patient. Given the force of injury involved, other organs may be affected, particularly the trachea, which may also be ruptured.

Clinically, the patient may complain of chest or abdominal pain; later, signs of sepsis from contamination of the mediastinum and pleura may be present. Chest radiographs may show widening of the mediastinum, subcutaneous emphysema, pneumothorax, hydrothorax, or a combination. Usually the lower esophagus is affected; rupture is therefore into the left pleural cavity. Tube thoracostomy for drainage is often performed. If the output is suggestive of gastric contents or the injury is otherwise clinically suspected, a contrast study or endoscopy of the upper GI tract is indicated to evaluate the patient for esophageal injury.

Treatment is usually surgical and should be performed early to minimize contamination of mediastinum and pleura. Primary repair versus diversion will depend on the condition of the esophagus, degree of contamination present, and, in some cases, the length of time from injury to surgery.

Thoracic duct injury and chylothorax: Injuries to the thoracic great vessels may be complicated by concomitant thoracic duct injury, which, if unrecognized, may produce devastating morbidity due to severe nutritional depletion. Initial management of a delayed chylothorax is always aggressive but nonoperative. Hyperalimentation with total enteral foodstuff restriction (i.e. parenteral hyperalimentation) may result in a significant number of spontaneously sealing thoracic duct injuries. Failure to spontaneously seal after 5-7 days indicates the need for surgical intervention, which should be individualized because the optimal approach is controversial. The duct could be approached either thoracoscopically or trough right thoracotomy aiming to ligate the duct as it traverses the diaphragm.

Empyema Thoracis

Empyema is a condition in which pus and fluid from infected tissue collects in a body cavity. The name comes from the Greek word *empyein* meaning pus-producing (suppurate). Empyema is most often used to refer to collections of pus in the space around the lungs (pleural cavity), but sometimes refers to similar collections in the gall bladder or the pelvic cavity.

Anatomic considerations: Within the thoracic cavity are 2 pleural cavities and the mediastinum. The mediastinum contains the heart, the esophagus, the trachea, the great vessels, and other structures. The left and right pleural cavities are situated lateral to the mediastinum and contain the lungs and their associated structures. Each lung is surrounded by a pleural sac. The pleura is a continuous layer of mesothelial cells and submesothelial matrix, which covers the chest wall and lung. The parietal pleura lines the wall of the pleural cavity and is attached by connective tissue to the internal thoracic wall and superior surface of the diaphragm. During respiration, the parietal pleura moves with the contraction of the diaphragm and expansion of the chest wall. The parietal pleura is continuous with the visceral pleura at the hilum where structures enter and leave the lung. The visceral pleura envelops the lung and is attached to the lung by connective tissue. Reflections of the visceral pleura line the lobes of the lungs and are visualized as the lung fissures on chest radiographs. Normally, the visceral pleural is in close proximity to the parietal pleura as the lung expands to fill the potential pleural space.

Physiologic consideration: The pleural space contains a minimal amount of fluid (approximately 5 ml in a typical 70-kg individual). The pleural fluid decreases friction between the surfaces. Sub pleural lymphatics drain excessive pleural fluid. Normally, the secretion of fluid into the pleural space is in equilibrium with drainage via the sub pleural lymphatics. The pleural lymphatic system is capable of draining almost 500 ml each day. When the ability of the lymphatics to drain the pleural space is exceeded, an effusion begins to form.

Pathology: Although any inflammatory condition within the vicinity of the pleura may lead to empyema (infected chest wall wound, osteomyelitis of ribs, post-thoracotomy, rupture or carcinoma of esophagus and subphrenic abscess, yet empyema is most

commonly a sequelae of pneumonia. The inflammatory response increases the permeability of the mesothelial cells present at the outer layer of the pleura. The increased permeability to albumin and other proteins explains why pleural effusions due to infection are rich in protein. The formation of an empyema has 3 stages:

1. Exudative stage: the inflammatory process increases the permeability of mesothelial cells, with the formation of a protein-rich fluid and brings more inflammatory cells. Characteristically, neutrophils that are normally absent in the pleural space become abundant and their number increases progressively and rapidly. Glucose and pH levels are normal. At this stage, drainage of the characteristically this “free-flowing” effusion and appropriate antimicrobial therapy is normally sufficient for treatment.
2. Fibrinolytic stage: the fluid becomes more viscous due to activation of coagulation factors and fibroblasts which begin coating the pleural membrane with the formation of an adhesive meshwork. Glucose and pH levels become lower than normal.
3. Organizing stage: Fibroblast activity causes adherence to the visceral and parietal pleura with the fluid being trapped inside formed loculations. This activity may progress with the formation of pleural peels in which the pleural layers are indistinguishable. Pus, which is a protein-rich fluid with inflammatory cells and debris, is present in the pleural space. Surgical intervention is often required at this stage.

Clinical picture: Acute empyema with acute fulminating toxemia, profound shock and pleural pain is now rare, except when it follows esophageal perforation or rupture of lung abscess. The patient usually presents with a subacute less severe form owing to the efficiency of the antibiotics administrated for the treatment of the primary condition. Clinical signs are those of a patient showing slow convalescence of the triggering illness (e.g. pneumonia) together with the appearance of stony dullness, absent breath sounds, diminished chest movement and other signs of pleural effusion. Empyema becomes chronic either due to the mismanagement or misdiagnosis of the acute or subacute stage or due to the persistence of the triggering illness in the lung (bronchiectesis, lung abscess, carcinoma) pleura (foreign body, mesothelioma), or chest wall (rib sequestrum). Toxic symptoms are replaced by vague illness, anemia, low grade fever, loss of appetite, chronic dyspnea, etc…

In the course of pneumonia, and before the modern era of antibiotics, most effusions and empyemas were related to *Streptococcus pneumoniae* pneumonia. Pneumococcal infections normally respond to antibiotic therapy, and these are less frequently seen in association with pleural-space infections today. Staphylococcal species and anaerobic pathogens are now the most common microorganisms associated with empyema. Because half of staphylococcal effusions progress to empyema, early drainage may be indicated if this organism is isolated. The re-emergence of tuberculosis may result in an increased association of *Mycobacterium* species with empyema.

Most parapneumonic effusions resolve with appropriate and timely antibiotic therapy. However, other effusions can progress to an empyema if prompt drainage is not performed. Interventions are uncomfortable and potential complications are risks. In rare cases, an infected pleural collection can extend through the pleural space into the chest wall, called empyema necessitatis. A fluctuant mass can be palpable and if the empyema is left untreated, the infection can extend to the surface and drain spontaneously. This is considered a pleurocutaneous fistula. On the other hand, an empyema may discharge either continuously or intermittently into a bronchus and hence, considered as a bronchopleural fistula.

Diagnosis:

1- A standard 2-view (postero-anterior and lateral) chest x-ray remains the first study for evaluating effusion or empyema. Free-flowing pleural fluid collects in the dependent portion of the pleural space. On 2-view chest radiographs, pleural fluid obscures the costophrenic angle and approximately 75 ml of fluid is required to blunt the posterior costophrenic angle on a lateral chest radiograph. Almost 200 ml of fluid is required to blunt the lateral costophrenic angles on frontal radiographs. If loculations have formed, fluid opacity may be seen in a nondependent area.

2- Ultrasonography and CAT scan. Ultrasonography may show smaller volumes of pleural fluid and provide information on viscosity. Ultrasonography also may quickly demonstrate the presence or absence of septa within the pleural fluid collection. CT of the chest is the imaging study that provides the most information. CT imaging depicts fluid, loculation, and thickening of the pleural membranes. Gas bubbles within the pleural space are strongly suggestive of empyema in the proper clinical context (i.e. in

the absence of recent thoracentesis). Lung windows can demonstrate pneumonia adjacent to the abnormal pleural collection. Soft tissue windows can demonstrate a cause for the empyema, such as esophageal rupture or mediastinal surgery. CT and ultrasonography are also used for biopsy and, sometimes for the placement of drainage catheters.

3- Pleural taping is always indicated for diagnosis as well as for culture and sensitivity tests. Diagnostic aspiration is usually carried out with a wide bore needle in the 5^{th} -7^{th} spaces mid-axillary line and 7^{th} intercostal space infra scapular line, with care not to introduce air in the pleural cavity. Part of aspirate must be sent for culture and sensitivity and another part for pathological examination.

Treatment:

1- General measures include management of the cause, antibiotics according to culture and sensitivity tests and raising the general condition of the patient by healthy diet, tonics and vitamins. Respiratory exercises are a must to eliminate the dead space created after aspiration of the empyema fluid thus allowing full expansion of the affected lung.

2- Thoracocentesis: a single thoracentesis may be effective at the earliest stages of empyema formation. If the effusion recurs, placement of a chest tube or small-bore catheter for continuous drainage is the next step. The placement of a small-bore catheter (thin-walled 8F-to-16F catheter) is often more comfortable for the patient than the traditional thoracostomy tube and is best accomplished under ultrasonographic or CT guidance. A larger bore intercostal tube is however needed for the drainage of thickened pus. The intra pleural injection of fibrinolytics increases the success rate of catheter drainage by decreasing the viscosity of the effusion and dissolving some adhesions and fibrinolytic therapy can reduced the need for surgery. Aspiration should be repeated until fluid is no longer obtained and the x-rays show no residual opacity.

3- Surgical intervention is still required in effusions with multiple loculations that are difficult to drain and effusions that have not responded to catheter drainage. Empyema at the organizing stage requires surgical intervention too. Surgical interventions may include the following:

a) Thoracoscopic debridement
b) Video-assisted thoracoscopic surgery (VATS): This relatively new intervention has reduced the frequency of open surgery.
c) Open drainage and rib resection: the selected site of drainage lies immediately above the lowest limit of the empyema posteriorly which is the most dependent point when the patient sits up in bed. Through a short vertical incision over the selected rib, about 5-7 cm of it is resected subperiostally and the empyema sac is entered. Complete evacuation is attempted, the hole of entry is widened and a thick bore intercostal tube is placed for open drainage. Healing requires frequent dressing and shortening of the tube that is gradually pushed to the outside by a wound that heals from within outwards. Healing is usually complete between 6 to 8 weeks.
d) Open surgical decortication: This technique is the most invasive and time consuming but is required when thick pleural peels are present on the visceral pleura. Through a posterolateral thoracotomy, the pleural peel is totally removed leaving the lung and chest wall free to expand. Postoperatively, physiotherapy and respiratory exercises are mandatory to ensure full lung expansion.

The mediastinum

Anatomy considerations: The mediastinal borders are the thoracic inlet superiorly, the diaphragm inferiorly, the sternum anteriorly, the spine posteriorly and the pleurae on either sides laterally. The anterosuperior compartment is anterior to pericardium and contains the thymus and great vessels. The middle, or visceral, compartment is between anterior and posterior pericardial reflections and contains the heart, phrenic nerves, tracheal bifurcation, major bronchi and lymph nodes. The posterior, or paravertebral, compartment is posterior to posterior pericardial reflection and contains the esophagus, vagus nerves, sympathetic chains, thoracic duct, descending aorta, and azygos/hemiazygos venous system.

A. Mediastinal Emphysema follows the introduction of air from esophagus, tracheobronchial tree, neck, or abdomen into the mediastinum. Although the condition may rarely occur spontaneously, yet it most commonly follows a penetrating or blunt trauma. The patient presents with a substernal chest pain, crepitations, and pericardial crunching sound and cardiac tamponade may follow. Depending upon the cause, the

condition may resolve spontaneously or require chest tube placement for pneumothorax.

B. Mediastinitis occurs in about 1% of patients after median sternotomy for open heart surgery. Risk factors include prolonged surgery or cardiopulmonary bypass, re-exploration, wound dehiscence, shock, and use of bilateral internal mammary artery grafts in patients who are older or have diabetes. The condition presents as persistent fever and leucocytosis after surgery and is life threatening, unless properly managed. Best treatment is early intervention, reopening of the incision, effective mediastinal and sternal debridement and proper antibiotic therapy according to culture and sensitivity tests. In heavily infected or recurrent cases, healing could be promoted by the use of rectus or pectoralis muscles flaps for better blood supply to the healing mediastinal tissues.

C. Superior vena cava obstruction is usually of a malignant origin due to bronchogenic carcinoma, especially epidermoid type, and to less extent lymphomas. Benign causes account for 10% of the cases and are mostly iatrogenic due to pacemaker electrode, hyperalimentation or other central venous lines, but may be also inflammatory due to histoplasmosis, idiopathic fibrosing mediastinitis or as a part of Behcet's syndrome. Clinically, the patient presents with a swelling face, neck and arms. Shortness of breath, orthopnea, cough and chest pain suggest upper airway obstruction. Headaches, syncope and lethargy are caused by cerebral edema from venous hypertension and, classically, symptoms become worse when the patient bends forward. On the other hand, symptoms of cerebral or laryngeal edema are associated with a reduced life expectancy of about 6 weeks, demanding urgent intervention.

Diagnosis is acquired through chest x-ray that may demonstrate a right hilar mass in case of bronchogenic carcinoma, anterior mediastinal mass in case of lymphoma or calcification in case of histoplasmosis. Simultaneous bilateral arm venogram can define obstruction and collateral circulation and identify the presence of a thrombus. CAT scan chest is essential for the assessment of mediastinum, determine patency of jugular veins. Mediastinoscopy or CAT scan directed needle biopsy are sometimes needed to determine the cause. Differential diagnosis include benign diseases (TB, fungal infections, retrosternal thyroid, and aortic aneurysms) and mostly malignant neoplasms, including lymphoma, Hodgkin's disease, small cell carcinoma, squamous cell carcinoma, germ cell tumors, and breast cancer. Due to malignant predominance, nearly all patients receive radiation therapy. Although 80-90% of patients are

symptomatically relieved, 50% of patients relapse even in benign disease, as although collaterals develop, thrombosis will continue to propagate and occlude these collaterals over time. Medical therapy includes chemotherapy for lymphomas and bronchogenic small cell carcinoma, diuretics and corticosteroids to reduce cerebral edema, anticoagulants in selected patients to prevent thrombus propagation and thrombolytic therapy in selected patients presenting with acute thrombosis. Surgery is indicated in severe forms associated with thrombosis of caval tributaries and inadequate collateral circulation.

Superior vena cava bypass with composite autogenous vein or PTFE grafts are indicated for palliation. Through a cervico-sternotomy, one end of a PTFE Grafts (8-10 mm) is proximally anastomosed to internal jugular and the other end is distally anastomosed directly to the right atrium. Another way to bypass the SVC is to dissect the patient's long saphenous vein and cut its lower most end at the ankle, leaving the saphenofemoral junction intact. Through a long subcutaneous tunnel made in the chest, the vein is passed upwards and the cut end is then anastomosed to the jugular vein. Hence, the course of blood coming from the neck will be as follows: jugular vein----extra anatomic saphenous vein in the chest wall-----saphenofemoral junction-----femoral vein-----inferior vena cava and lastly, right atrium. Good results were achieved by either technique, especially in patients with Behcet's syndrome.

D. Mediastinal tumors

Most common tumors are: neurogenic (20%), thymomas (20%), primary cysts (20%), lymphomas (13%), and germ-cell tumors (10%). Neurogenic tumors and non-Hodgkin's lymphomas are the most common tumors in children. Regarding location, most mediastinal tumors are located in anterosuperior compartment (54%), followed by posterior (26%) and middle (20%) tumors. Regarding malignancy, a significant portion (25-40%) of mediastinal tumors is malignant; especially tumors in anterosuperior mediastinum as well as tumors in patients between the ages of 10 and 40.

Tumors and Cysts by Location		
Anterior	**Middle**	**Posterior**
Thymoma Germ cell tumor Lymphoma Hemangioma Parathyroid adenoma Thymic cyst	Enterogenous cyst Mesothelial cyst Lymphoma Thoracic duct cyst Granuloma Hamartoma	Neurogenic origin Neurenteric cyst Lymphoma

Lipoma Aberrant thyroid Lymphangioma		

Clinically, about two-thirds of patients will have symptoms at the time of diagnosis and the absence of symptoms is a reasonably good indicator that a diagnosed tumor is benign. Most common symptoms include chest pain, cough, and fever. Signs of mechanical compression or invasion of mediastinal structures are more common with malignant tumors. Paraneoplastic syndromes are not uncommon and include Cushing's syndrome, thyrotoxicosis, hypertension, hypercalcemia, hypoglycemia, diarrhea, and gynecomastia.

Although chest x-ray can localize the tumor and give information on calcification and relative density of the tumor, yet CAT scanning identifies chest wall invasion, multiple masses, and extension into spinal column. MRI is more accurate for vascular involvement and intracardiac pathology. Echocardiography is useful for patients with middle compartment tumors to localize between intracardiac and pericardial tumors. Guided needle biopsy can make a diagnosis of malignancy in 80-90% of patients. Lastly, Mediastinoscopy or even mediastinotomy are sometimes needed to make a diagnosis and establish respectability.

A. Thymoma represents 20% of all mediastinal masses in adults with a peak incidence in 3rd to 5th decades of life; rare in children. About half are of mixed cell type, followed by epithelial (28%) and lymphocytic (20%) types. Between 15 and 65% of thymomas are benign and are frequently associated with paraneoplastic syndrome, most commonly myasthenia gravis. As a rule, Myasthenia gravis is diagnosed in 30-50% of patients with a thymoma, and 15% of myasthenia patients will have a thymoma. Prognosis is dependent on stage of tumor, and not on presence of myasthenia gravis. Surgery involves the removal of all anterior mediastinal tissue, including involved lung, pleura, pericardium, and SVC/ innominate vein. In addition, patients with stage IIa or higher disease should receive postoperative radiation. At 5 years after resection, 25-30% of patients will have complete resolution of myasthenia symptoms and 30-50% will be improved. Chemotherapy is indicated for stage III or IV disease; otherwise tumor debulking (i.e. removal of as much as one can safely do) may offer some relief even in the absence of evidence for increased survival.

B. Thymic Carcinoid originates from Kulchitsky cells in the thymus especially in males, however not associated with myasthenia gravis or the carcinoid syndrome. On

the other hand, the tumor may cause other paraneoplastic syndromes –especially Cushing's syndrome- denoting a very poor prognosis. Up to 75% of patients will develop local recurrence or metastases and the overall cure rate is low with a mean survival of 3 years.

C. Lymphoma: While only 5-10% of lymphoma patients will have isolated mediastinal disease that is usually symptomatic; 40-70% of patients with lymphoma will have mediastinal involvement during their disease course. Nodular sclerosing and lymphocyte predominance forms of Hodgkin's lymphoma are the most common to cause mediastinal involvement, followed by lymphoblastic non-Hodgkin's lymphoma. Surgery is indicated if fine-needle aspiration is inconclusive or to evaluate residual mass after chemotherapy. Surgical options include cervical mediastinoscopy, parasternal mediastinotomy, and thoracoscopy.

D. Germ Cell Tumors comprise 15-25% of anterior mediastinal masses and are most common in children and young adults. Germ cell tumors include teratomas, teratocarcinomas, seminomas, embryonal cell carcinomas, choriocarcinomas, and endodermal cell or yolk-sac tumors. About 60% are benign and 40% are malignant:

Predominantly Benign Tumors: Teratomas are complex multiple tissue element tumors that produce symptoms only by mechanical effects. The simplest form is the **dermoid cyst**, which consists of mostly dermal and epidermal tissue. More complex teratomas may have well-differentiated bone, cartilage, nerve, or glandular tissue.

Malignant Tumors have a male predominance and are mostly symptomatic. Forty percent are seminomas and 60% are non-seminomas (embryonal cell, choriocarcinoma, yolk-sac, and teratocarcinoma). Initial surgical intervention is typically performed only for diagnosis, due to the high radiosensitivity of seminomas and frequent metastatic disease in non-seminomas (Cis-platinum chemotherapy). Surgical resection after induction of chemotherapy may have a role in non-seminomatous tumors and the overall 5 year survival varies between 50 and 80%.

E. Endocrine Tumors: Eighty percent of intra thoracic thyroid tumors are substernal extensions of a cervical goiter and the true intra thoracic thyroid, which derives blood supply from thoracic vessels, comprises only 1% of all mediastinal tumors. They are more common in women in their 6th to 7th decade of life and most are adenomas. Clinically, they usually present with tracheal or esophageal compression and thyrotoxicosis is uncommon. Iodine 131 scanning should be done to identify the presence of functioning cervical thyroid tissue before resecting these tumors either

through a cervical incision for substernal extensions or through a limited median sternotomy for true intra thoracic lesions.

Most parathyroid tumors are adenomas and are found by the superior pole of the thymus due to common embryogenesis from the third branchial cleft. Symptoms are usually due to hyperparathyroid syndrome but parathyroid cysts are not usually hormonally active.

F. Primary Cysts: Bronchogenic cysts are the most common primary cysts in the mediastinum (5%) that arise from ventral foregut and are usually located in the subcarinal or right paratracheal region. Two-thirds are asymptomatic and symptoms include tracheobroncial or esophageal compression and infection from tracheobronchial communication. Complete excision is recommended, even if asymptomatic, to prevent late complications

Esophageal/Enteric cysts comprise 3-5% of mediastinal tumors, are more common in children and tend to occur in the lower third of the esophagus. Dysphagia is the most common symptom. CT scanning is essential in patients with vertebral anomalies to evaluate for possible spinal cord involvement (neuroenteric cyst) and endoscopic biopsy should be avoided, as this may cause cyst perforation and infection. Complete excision is indicated. The less invasive thoracoscopic approach can be used for uninfected cysts.

Pleuropericardial cysts are uncommon. Classically, they occur at the pericardiophrenic angles, 70-80% on the right side and are usually asymptomatic. Guided needle aspiration is the initial therapy of choice and surgical excision is indicated if the cyst recurs or if the diagnosis is in doubt.

G. Neurogenic Tumors: Most posterior mediastinal masses are of neurogenic origin. In adults, 95% of these tumors are benign and are usually asymptomatic. In children however, most neurogenic tumors are malignant. Classified according to cell origin; most arise from intercostal nerve or sympathetic chain. Neurilemomas are the most common and originate from Schwann's cells, while Neurofibromas originate from peripheral nerve and both types of tumors are associated with von Recklinghausen's disease. Chest CT is sufficient for diagnosis of most of these tumors, and MRI should be used when an intraspinal component is present.

Intercostal nerve	**Sympathetic ganglia**	**Paraganglia cells**
Neurofibroma Neurilemoma	Ganglioma Ganglioneuroblastoma	Paraganglioma pheochromcytoma

Neurofibrosarcoma	Neuroblastoma	

Benign tumors (neurofibroma, neurilemoma, ganglioneuroma) can be effectively treated with local excision. Combined thoracic and neurosurgical approach is indicated for tumors with intraspinal extension. While recurrence is rare for benign tumors, local recurrence is common for malignant tumors and the overall prognosis is poor.

Surgical treatment of bronchogenic carcinoma

Histological consideration: Four histological types are distinguished: squamous cell, commonly arising in the larger bronchi and spreading by direct extension and lymph node metastasis; undifferentiated small cell, often associated with early hematogenous metastases; undifferentiated large cell, usually spreading through the bloodstream; and adenocarcinoma, commonly peripheral, often spreading through the bloodstream. All types also commonly spread via the lymphatics. Bronchioloalveolar carcinoma, a subtype of adenocarcinoma, consolidates airspaces and often does not extend beyond the lungs.

Staging of bronchogenic carcinoma: Staging lung cancer is useful in prognosis as well as in selection of treatment. Although may be done clinically but is more accurate with procedures that yield information about the extent of local and systemic disease including; CT chest and abdomen or brain, bone scan as well as invasive bronchoscopy, mediastinoscopy, and even exploratory thoracotomy. Nevertheless, the TNM (tumor, node, metastasis) system is the standard staging classification for non-small cell carcinoma. On the other hand, small cell carcinoma has usually metastasized by the time it is diagnosed and is staged as either limited (confined to one hemithorax with or without involvement of mediastinal and ipsilateral supraclavicular lymph nodes) or extensive (spread beyond this point).

Preoperative evaluation: Although the surgeon’s best hope is to know both: tumor staging and histological types before surgery, yet this is not always feasible. In practice, the histological type of many patients can only be made intraoperatively with frozen section or even postoperatively, after the definitive pathological examination of the resected tumor. Moreover, preoperative staging is also revised during surgery, which further determines resectability.

As pneumonectomy (removal of the whole lung) is either preoperatively indicated from the start or becomes an intra operative decision facing a more extensive lesion than expected and hence, pulmonary function tests must be performed before surgery. The functional criteria for pneumonectomy (i.e. patient can live comfortably with only one lung after surgery) are: 1) normal Pa_{CO2} 2) a forced expiratory volume in 1 sec (FEV_1) of more than 2 liters and 3) is equal to more than 50% of the observed forced vital capacity.

Surgical indications: surgery is the treatment of choice for non-small cell carcinoma; however, and as previously mentioned, histological diagnosis is not always made preoperatively. Nevertheless, surgery is indicated in stages I and II as well as class T3N0M0 or T3N1M0 patients (because of chest wall invasion). The type of surgery required depends on the site of lesion as well as the extent of local invasion: While removal of the affected lobe (lobectomy) can be sufficient for peripheral lesions, pneumonectomy is usually required for central endo bronchial lesions and is supplemented with en bloc chest wall removal, in case of chest wall invasion.

Results of surgery: In general, and although up to 1/3 of tumors are resactable, the overall 5 years survival is no more than 13%. Depending upon the histological type and tumor stage, this rate however is raised to up to 70% after resection of stage I non-small cell carcinoma to reach more than 88% after resection of peripheral nodular lesions. On the other hand, and because small cell carcinoma has almost always spread beyond the primary site at the time of diagnosis, it is either inoperable or irresectable or shows as high as 25-50% recurrence after primary resection and adjuvant chemotherapy.

Improved survival has being reported when preoperative irradiation or neoadjuvant chemotherapy were used in Pancoast tumors as well as in stages II, IIIA, and IIIB non-small cell carcinoma; respectively.

Role of chemotherapy and radiotherapy: In brief, chemotherapy with multiple drugs, particularly cisplatin and topoisomerase inhibitors are used for all patients with small cell carcinoma as well as unresectable stage IIIA, IIIB, or IV non-small cell carcinomas. The role of radiotherapy is however of palliative nature for relief of obstruction induced by endo bronchial lesion, compression on SVC or spinal cord and for control of hemoptysis or brain metastasis.

Basic principles of Open Heart Surgery And Cardiopulmonary Bypass

Operative treatment of cardiac abnormalities is advancing rapidly, particularly with the advent of new techniques for controlling the circulation and myocardial preservation during surgery. The procedures can be, for practical purposes, placed in one of 4 main categories:

A- Extra cardiac operations carried out on the main vessels outside the heart as well as the pericardium. Examples include pericardiectomy for constrictive pericarditis, ligation of patent ductus arteriosus, repair of aortic coarctation and various palliative surgeries for congenital cyanotic heart diseases. Surgery is usually performed without cardiopulmonary bypass and hence, avoids its inherent complications. Also avoided is the prolonged mechanical ventilation as most of those patients are extubated on table after surgery. After surgery, the ICU stay is either short or the patient is directly admitted in an intermediate care facility.

B- Closed cardiac operations in which the surgeon tried to blindly repair an intra cardiac pathology whether by finger or instrument placed inside the heart through a purse string suture to avoid blood loss are no more than history. The last procedure that was still carried out in some centers 10 years ago was closed mitral valvotomy and is actually replaced by balloon valvotomy carried out by invasive cardiologists. In fact, most of today cardiac surgery is performed by open heart surgery techniques.

C- Classical Open heart surgery: In order to open the cardiac chambers and perform a decent reliable and safe cardiac surgery, the heart must be empty and motionless so as to see and be able to operate with precision. The heart should be functionally disconnected form the circulation so as no blood is coming back inside the heart from the vena cava and pulmonary veins, and no air is going out form the opened-to-air heart to embolize other body organs. The question was the following: If the heart has to be practically out of the circulation (i.e. arrested, not receiving or pumping blood), we will be in need of a machine that can carry out safely the cardiac function (receiving and pumping blood from and to the circulation); and it was Charles Gibbons' who invented the first safe cardiopulmonary by pass (CPB) machine in conjunction with IBM engineers in 1954. Despite of hundreds of renovations made on such machines, the basic idea was and remained simple (figure 5):

1- Both right and left sides of the heart must be empty during surgery and hence, systemic venous blood is drained by a cannula placed in the right atrium to the CPB machine. The latter will then pump back the blood into the systemic circulation through another cannula placed in the ascending aorta.

2- It is evident that the collected venous blood needs to be oxygenated before being pumped back to the patient's ascending aorta and systemic circulation and hence, a synthetic bio membrane oxygenator has to be incorporated within the CPB circuit to oxygenate the drained venous blood.

3- It is also evident that the patient's blood can thrombose when collected and pumped by a series of foreign synthetic materials and surfaces and hence, the patient must be fully heparinized during CPB. After CPB, and in order to achieve postoperative hemostasis, heparin is reversed by protamine sulfate on 1 to 1 basis.

4- In conclusion both the heart and lungs are functionally bypassed during CPB and their main work which is: to receive and pump back blood i.e. to maintain the systemic circulation (heart function) and to oxygenate this blood (lungs function) is carried out by the CPB machine.

5- After establishing CPB as described, the heart can be arrested and respiration can be stopped without fear of other organ damage. The question is now the following: how would a heart being arrested and deprived from the circulation survive the period of surgery? The absence of a perfect method to protect the heart during this period explains the various measures adopted for myocardial preservation. Classically, the ascending aorta is cross-clamped and a cold (4 C) crystalloid hyperkalaemic solution is infused through the aortic root into the coronary circulation. The solution arrests the heart during diastole and keep it arrested (potassium), brings down to minimum its metabolic needs (coldness) and combats acidosis induced by ischemia (different types of added buffer). The infusion is then repeated every half an hour, until intracardiac procedures are terminated and cardiac arrest is no more needed. Another common way of myocardial protection is to infuse cold hyperkalaemic blood and not crystalloid into the coronary circulation. Besides the above mentioned protective effects, the presence of blood add nourishment to the myocardium during the arrest and is of course the best known buffer to fight arrest induced acidosis. Nevertheless, Besides the use of one of the many types of cardioplegia, other procedures that are concomitantly carried out to improve myocardial preservation are: shortening the procedure and hence, myocardial ischemic time, lowering the temperature of CPB itself (around 28 C) and covering the

heart itself with ice cold saline which further drops the myocardial temperature and hence, minimizing its metabolic demands.

6- After terminating the intra cardiac procedure (heart valve replacement or repair, coronary artery bypass grafting, repair of congenital anomaly, etc…) all the carried out processes are simply reversed: the cardiac chamber is closed and carefully de-aired so that no air embolism occurs when the ascending aorta is de-clamped and the heart is reconnected to the circulation. Ice cold saline is removed and CPB blood is re-warmed to normal temperature. The patient is gradually weaned off CPB by allowing the heart to gradually receive and pump more blood until it regains its full action. After resuming normal ventilation and ensuring that the heart can alone ensure full hemodynamic stability, cannulae are removed, heparin is reversed by protamine sulfate, hemostasis is carried out and the wound is closed before discharging the classically anesthetized and ventilated patient to the ICU.

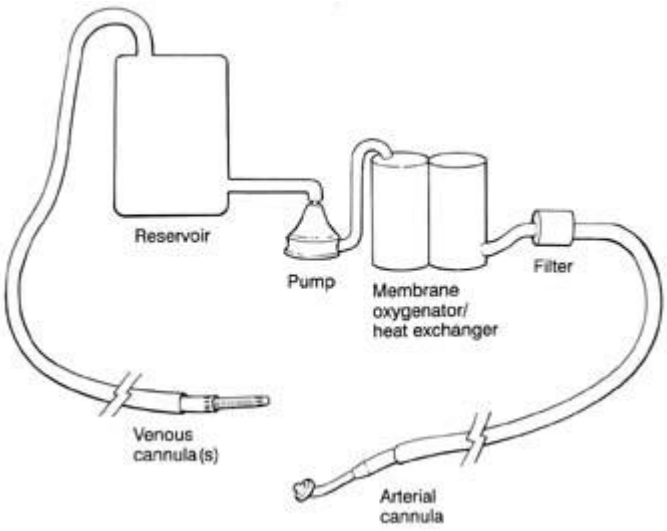

Figure 5: The figure shows the basic cardiopulmonary bypass circuit. Blood is collected from the right atrium via the venous cannula (on the left). By gravity blood is then collected in a venous reservoir placed at a lower level than the operating table. The electric roller pump receives the blood from the reservoir to actively push it inside the membrane oxygenator. The pump controls the rate by which the oxygenated blood is then pushed back to the systemic circulation via the cannula placed inside the ascending aorta and hence, controls the cardiac output during bypass. As shown, the system is further supplied by a heat exchanger to control the blood, and hence, the

body temperature and a filter to catch any formed debris or air bubbles.

D-Minimal invasive cardiac surgery:

Cardiac surgery is a rapidly progressing branch for being essentially based on the fast evolving modern technology. The field of progress is vast and ranges from robotic surgery and genetic remodeling of pigs' hearts to prevent their rejection once transplanted in man to automated anastomosis equipments that replaced the surgeon's talent in creating a hand made anastomosis. The aim is to permit a safer surgery with a short hospital stay and a better short as well as long term prognosis. In this setting, minimal invasive cardiac surgery includes a variety of procedures which aim to decrease the inherent morbidity of open heart surgery. Their use is both patient and surgeon dependent and the gained benefit must be always weighted with the possible drawbacks. The most famous examples are the following:

1- Minimal invasive approach: classically open heart surgery is performed through median sternotomy which provides an excellent exposure and almost permits all known open heart procedures. However, the scar of median sternotomy is always visible, sometimes ugly and depressing especially for young adults. Although less painful than a thoracotomy, yet delayed healing is associated with severe pains that can last for months. Healing problems are met with in obese and diabetic patients as well as in those cases where both mammary arteries (a major blood source for the sternum) are used for coronary artery bypass grafting. Moreover and whenever infection settles in, the mediastinum can be involved with a resulting -sometimes fatal- mediastinitis. A limited left thoracotomy (5 cm length) can be used for bypassing a stenosed left anterior descending coronary artery (major and most important coronary artery) by an internal mammary artery that is harvested endoscopically. The anastomosis can be carried out endoscopically too, which can limit incisions to 3 short hardly visible ones. Equally, many open heart procedures (mitral valve replacement, closure of atrial septal defect, etc…) can be carried out through a limited practically invisible sub-mammary right thoracotomy. Although minimal invasive approaches have the advantages of giving a better and rapidly healing scar, yet they have the disadvantages of needing more experienced surgeons for the small field they supply, of being insufficient in cases of emergency and of not allowing performing all types of surgeries.

2- Coronary artery bypass grafting with or without the use of CPB. Although arresting the heart is mandatory for all intra-cardiac procedures, yet coronary artery bypass grafting (CABG) does not involve opening the heart itself, as coronary arteries just run on the heart surface and hence, some surgeons prefer to carry CABG on beating heart with or without CPB. The procedure is tempting as the heart is not arrested and hence the arrest-induced myocardial ischemia is avoided however the perfection of coronary anastomosis while the heart is beating can always be questionable. Avoiding CPB offers the advantages of avoiding its hazards which includes: postoperative bleeding due to coagulation defects, CNS complications due to cerebral edema and air embolism etc… In absence of CPB, there is no machine to support the circulation during surgery and the heart remains the sole generator of body perfusion. As surgery on the beating heart necessitates cardiac manipulations that can induce serious arrhythmias and hemodynamic instability, a not less than perfect anesthetic techniques are required.

Coronary Artery Bypass Grafting

Stenotic atherosclerotic coronary artery disease is narrowing of the coronary arteries caused by atherosclerosis, which when sufficiently severe, limits the flow of blood to myocardium. At first, there is a limitation of the coronary flow reserve and hence, the patient experience chest pain when needing such reserve i.e. on effort. With the increase in stenosis, the normal coronary blood flow is compromised with the patient experiencing angina at rest. With worsening of the condition, the coronary artery is occluded which if sudden and in the absence of sufficient collaterals; myocardial infarction supervenes. It is worth noting, that this gradual progression from angina at effort to angina at rest to myocardial infarction is not a must and many patients may present, for the first time, with unstable angina or even fatal myocardial infarction.

The resting ECG may be normal in the presence of a good myocardial flow reserve and in the absence of infarction. Although the diagnosis can be made with a treadmill test (ECG with effort), routine or dobutamine echocardiography and myocardial thallium uptake; yet cardiac catheterization for coronary arteriography remains the key element for assessment of the individual artery involvement, the degree of stenosis and, most importantly, whether and how urgent surgery or balloon angioplasty are indicated. In this setting, a 67% decrease in vessel diameter on

arteriography is equivalent to a 90% loss of arterial cross-sectional area and such a severe significant lesion is an indication for interference.

Anatomic considerations: From the surgeon standpoint, the coronary artery system is divided into 4 parts: the left main coronary artery, the left anterior descending coronary artery (and its diagonal branches), the circumflex coronary artery (and its marginal branches) and the right coronary artery (and its posterior descending branch). A significant lesion (or lesions) affecting one of the last three vessels (or one of their large branches) is considered as a one-vessel disease. The presence of significant lesions in 2 of those vessels (e.g. left anterior descending and right coronary artery) or their large branches is considered as a two-vessel disease and the affection of all 3 vessels is considered as a three-vessel disease. Although the presence of a significant lesion in the left main coronary artery jeopardizes both left anterior descending and circumflex coronary artery, yet it cannot be simply categorized as a two-vessel disease. In the former, only one thrombus can occlude both vessels while in the latter, 2 thrombosis events (and hence with less chance to happen) are needed for this catastrophic occlusion. In fact, a significant left main lesion is a surgical risk, balloon angioplasty is never attempted and surgery is urgently indicated.

Basic surgical concepts: A significantly severe coronary artery stenosis (67% lesion on arteriography or more) jeopardizes blood supply distal to the lesion and, as evident from its nomenclature, coronary artery bypass grafting (CABG) involves bypassing such a lesion to ensure myocardial blood flow. The left (or right) internal thoracic (mammary) artery can be easily dissected from the under surface of the chest wall and the cut distal end is then anastomosed to the involved stenotic coronary artery through a small (4-6 mm) arteriotomy made distal to the lesion. A significant lesion in the left anterior descending coronary artery (LAD) is best bypassed by the left internal mammary artery (LIMA) and, in fact, LIMA to LAD provides the best results in CABG with a 10 years patency rate of more than 90% (figure 6). Significant lesions affecting other arteries are usually bypassed by the use of either the radial artery resected from the non-dominant hand or the long saphenous vein of the leg. The conduit is at first implanted on the coronary arteriotomy made distal to the lesion (distal anastomosis) and then on the ascending aorta (proximal anastomosis); (figure 6). Other less commonly used conduits include: the right internal thoracic artery and the inferior epigastric artery.

As with any vascular anastomosis, the degree of blood flow is largely dependent on the presence of a good distal run-off. The presence of other lesions distal to the one

being bypassed increases the pressure against which blood will flow and condemns the formed anastomosis for an early or even catastrophic closure. Moreover, and besides the presence of a good distal run-off, the supplied myocardium should be still viable as there is no point in irrigating a dead muscle. In case of doubt myocardial viability is tested preoperatively either by thallium scanning or dobutamine echocardiography.

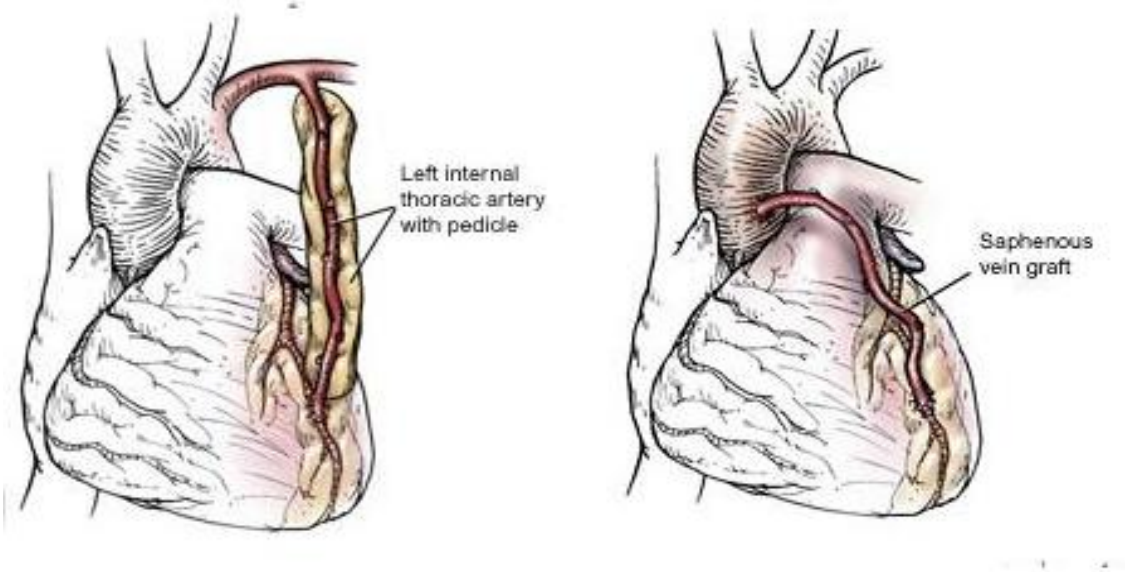

Figure 6: left internal mammary artery (LIMA) anastomosed to left anterior descending coronary artery (LAD) (left picture) and saphenous vein graft anastomosed proximally to the ascending aorta and distally to the LAD (right picture)

Indications of CABG: CABG is indicated for the relief of symptoms and improved quality of life and to reduce the incidence of subsequent nonfatal as well as fatal events such as myocardial infarction. The bases for the indications for CABG are the comparative benefits of the operation relative to those of no treatment, medical therapy and percutaneous coronary angioplasty (PTCA). The patient's age, sex, presence of diabetes or hypertension, angina class denoting the severity of patients' symptoms, the presence of a left main lesion and the number of affected vessels, the left ventricular function and myocardial viability; are all factors that should be carefully studied preoperatively. In short, CABG is indicated in patients with even less than significant (50%) left main coronary artery lesion even in the absence of symptoms for the high risk of sudden death associated with such cases. While patients with three-vessel disease as well as those presenting with two-vessel disease and not amenable to PTCA are candidates for surgery, patients with a single-vessel disease usually respond well to medical treatment or PTCA.

Results of CABG The contemporary results of coronary bypass surgery are excellent, with overall mortality under 3 percent. The 5-year survival is 88% and 10-year survival 75%. The maximal exercise capacity is improved, particularly when complete revascularization has been performed. About 95% of patients are free from symptoms at 5 years and the 10-year freedom is greater than 60%. Unfortunately, the proportion of high-risk patients presenting for surgery is increasing with time. Moreover, the presence of left main lesion, bad distal run-off with or without the need to perform endarterectomy, 30% or lower ejection fraction, need for repair of post-infarction VSD or mitral valve incompetence as well as resection of post-infarction ventricular aneurysm; each is a major risk factor for postoperative mortality and morbidity.

Heart valve replacement

Although the etiology of heart valves lesions can be congenital, traumatic, degenerative, inflammatory or even neoplastic; yet the endemic rheumatic heart disease is still by far the commonest cardiac pathology in Egypt resulting in valvular stenosis, incompetence or both. The commonest affected valve is the mitral valve followed by the aortic valve. Rheumatic tricuspid valve lesions are rare and only few cases of rheumatic pulmonary valve disease were reported world wide.

Rheumatic fever is an acute, often recurrent, inflammatory disease that generally follows a pharyngeal (but not skin) infection with group “A” beta-hemolytic streptococci, principally in children. Evidence strongly suggests that rheumatic fever is the result of an immune response to streptococcal antigens, inciting either a cross reaction to tissue antigens, or a streptococcal-induced autoimmune reaction to normal tissue antigens. Acute rheumatic fever is purely a medical condition.

The cardiac surgical implications of rheumatic fever primarily relate to the chronic stage of rheumatic heart disease, characterized principally by chronic, progressive, deforming valvular disease, that produces permanent dysfunction and severe, sometimes fatal, cardiac failure decades later. Chronic rheumatic valve disease is characterized by fibrous or fibrocalcific distortion of leaflets or cusps and valve commissures. In the mitral valve, chordal as well as papillary muscle involvement leads to additional deformities. Stenosis results from leaflet and chordal fibrous thickening and from commissural and chordal fusion, with or without secondary calcification. Regurgitation entails other mechanisms, including scarring-induced retraction of chordae and leaflets and, less commonly, fusion of a commissure in an opened

position. Combinations of lesions may yield valves that are both stenotic and regurgitant.

Diagnosis: Trans-thoracic echocardiography is the main tool for diagnosis and assessment of severity of valvular heart disease. Trans-esophageal echocardiography (TEE) is usually needed for the detection of associated thrombotic or vegetative deposits and is a helpful tool in diagnosing malfunctioning prosthetic cardiac valves. Cardiac catheterization is only indicated for the detection of associated coronary artery disease in patients above 40 years of age.

Surgical indications: Cardiac vale replacement (or repair) is mainly indicated for the hemodynamic disturbances produced by the valvular pathology. In isolated mitral stenosis, surgery is absolutely indicated in severe cases where echocardiography, will show an effective orifice area of 1 cm or less (normally 4 cm). Surgery is also indicated in patients with moderate mitral stenosis that experience paroxysmal nocturnal dyspnea or orthopnea despite of adequate medical treatment. The decision making in patients with mitral incompetence is some how less sharp, for the late development of symptoms despite of a long standing disease and the development of partially reversible or even irreversible left ventricular enlargement. Surgery is indicated in patients in NYHA class III or IV, NYHA class I or II but with a significant left ventricular enlargement of grade 3 or more on echocardiographic examination (on a scale of 1-6) as well as those symptomatic patients despite of adequate medical therapy.

An isolated stenotic aortic valve has to be surgically treated when it generates a gradient of 50 mm Hg or more across the left ventricular outflow tract, in an often symptomatic patient. A moderate to severe aortic incompetence usually produces progressive left ventricular dilatation and surgery is indicated whenever the cardiothoracic ratio exceeds 55%, even in asymptomatic patients. As the development of symptoms is usually late until the left ventricle has usually become very large, all symptomatic patients with aortic incompetence have to be dealt with surgically.

Outside hemodynamic measurements, surgery is indicated for valvular endocarditis that failed to respond to intensive medical therapy and in the case of a thromboembolic event suspected to originate on top of a valvular pathology (e.g. mitral stenosis). In the latter, however, surgery is usually deferred after the complete

resolution of the acute thromboembolic event for the significant cerebrovascular morbidity of open heart surgery carried out in patients with acute stroke.

Valve Reconstruction versus valve replacement: The major advantages of repair over replacement relate to the elimination of both prosthesis-related complications and the need for chronic anticoagulation. Other reported advantages include lower hospital mortality, better long-term function and a lower rate of postoperative endocarditis. Reconstructive techniques for mitral valve stenosis and/or incompetence however, are more easily applied to mitral valves with non-rheumatic disease. Reconstructive therapy of selected patients with aortic insufficiency and aortic dilatation may also be done but repair of aortic stenosis has been notably less successful.

Choice of the implanted prosthesis: Cardiac valvular substitutes are of two generic types, mechanical and biological tissue (figure 7). Prostheses function passively, responding to pressure and flow changes within the heart. The most widely used mechanical valves are composed of non-physiologic biomaterials that employ a rigid, mobile two pyrolytic carbon hemi disks in a carbon housing (e.g. St. Jude valve, Carbomedics valve, ATS valve). Pyrolytic carbon has high strength as well as fatigue and wear resistance; with exceptional biocompatibility, including thromboresistence.

In contrast, tissue valves are, to a large extent, anatomically similar to natural valves. The major advantages of tissue valves compared to mechanical prostheses are their pseudo-anatomic central flow and relative non-thrombogenicity, usually not requiring anticoagulant therapy. The most widely used bioprosthesis are xenografts, homografts and autografts. Xenografts are usually made from a porcine valve or bovine pericardium. Comparatively, aortic or pulmonary valves transplanted from one individual to another (homografts) have exceptionally good hemodynamic profiles, a low incidence of thromboembolic complications without chronic anticoagulation, and a low re-infection rate which makes homografts an excellent valve substitute in case of endocarditis. In children and young adults, the patient’s pulmonary valve is used as an autograft to replace his diseased aortic valve and a pulmonary homograft is then used to replace the native pulmonary valve. Although the procedure is complicated, yet it is highly indicated to permit future growth of the autograft proportional to the somatic growth of the patient.

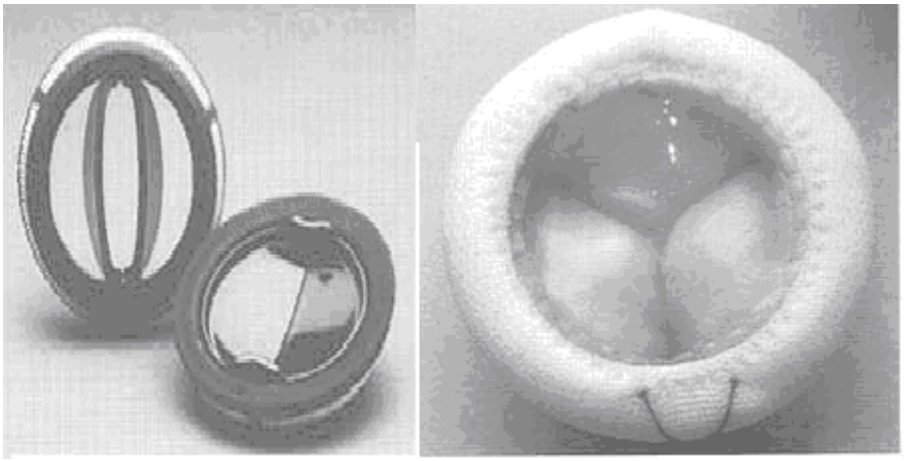

Figure 7: A bileaflet mechanical cardiac valve is seeing on the left; with a circular sewing ring enclosing a bileaflet mechanical occluder in the open (upper) as well as in the closed (lower) position. A biological cardiac valve is seeing on the right; with a circular sewing ring that encloses a porcine trileaflet aortic valve. After removal of the patient's native valve, the prosthetic valve ring is sewed to the patient's native valve ring with either continuous or interrupted sutures.

In general, a mechanical cardiac valve is the prosthesis of choice in most of the patients, for the expected high durability. Xenografts are indicated in old patients (> 60 years of age) as well as other patients in whom oral anticoagulation is not desired. On the contrary, their use is not advised in children as well as in other patients with a high rate of calcium turn-over, for early calcification and hence structural failure. If available, homografts are the best substitute for patients presenting with aortic valve endocarditis, for the eradication of infection and pulmonary autografts are the best choice for children with aortic valve disease, for the ability to grow in concordance with the expected somatic growth.

Results and complications of cardiac valve replacement: Early mortality after cardiac valve replacement is generally in the range of 3–5 %, the probability of 5-year survival is about 80% and of 10-year survival about 70%. On the other hand, accepted and non-preventable complications of prosthetic cardiac valves include: prosthetic valve endocarditis (PVE), prosthetic valve thrombosis (PVT) and structural failure necessitating re operation; now accounting for approximately 5–15 percent of all valve procedures and are associated with as much as 10-50% hospital mortality and hence,

are better avoided. Other uncommon complications necessitating prosthetic valve replacement are dehiscence of the prosthesis from the sewing ring creating areas of para-prosthetic leaks that result in significant regurgitation. Significant anemia can also develop due to the turbulence created in the vicinity of the leaky areas. Turbulence waves just hit and deform (but do not destruct) the red blood cells and hence, favors their early uptake by the reticuloendothelial system. With time, the only way to stop the developing anemia is to treat the cause; which is to change the prosthesis.

Prosthetic valve-related thromboembolism: The formation of a thrombus in relation to a mechanical cardiac valve is not only related to the presence of a foreign material (the prosthesis) in the circulation, but is also due to the design of the prosthesis itself which creates concomitant areas of turbulence and stasis; both of which invites thrombosis. Consequently, patients with mechanical cardiac valves are condemned for a lifelong anticoagulation therapy. In contrast, patients with bioprostheses are only anticoagulated for 6 months until the bioprosthetic sewing ring is invaded and covered by the host tissues; unless they have atrial fibrillation or another specific indication. The rate of thromboembolism in patients with mechanical valves is 2–4% per year. As chronic oral anticoagulation also induces a risk of hemorrhage, the optimal intensity of anticoagulation in patients with mechanical heart valves (balancing both thromboembolism and bleeding) both at approximately 2% per year is best achieved with a target INR (international normalized ratio) of 2.5-3.5. View the role played by platelet aggregation in the formation of both intracardiac and arterial thrombus (role that is not played in the formation of venous thrombosis), the addition of antiplatelets aggregates (persantin 75 mg three times daily) to oral anticoagulants allow to target a lower INR between 2 and 2.5 and is associated with less incidence of thromboembolic as well as bleeding events; compared to oral anticoagulants alone. In this setting however, the use of aspirin has been associated with significant bleeding and is better avoided. PVT is a surgical emergency, patient develop rapid progressive dyspnea with muffing of mechanical valve clicks. Congestive heart failure rapidly settles down and many patients have to be mechanically ventilated, with the need for positive inotropic support and the development of anuria. Every effort should be done to operate upon the patient before reaching this stage which carries more than 50% hospital mortality.

Patients in whom dental as well as other surgical procedure are indicated have 2 choices: either to be operated upon without change of anticoagulation regimen, as long as their target INR is within the therapeutic range. Experience has shown that although these patients will bleed more than others, yet no major postoperative bleeding occurs as long as a careful hemostasis is achieved. The other strategy is to heparinize those patients first with a loading dose of 70 IU/kg, followed by continuous IVI of heparin in regular saline given by a syringe pump so as to achieve a target PTT of 2 to 2.5 times normal. At this point, oral anticoagulants can be stopped, followed by stoppage of heparin infusion itself 6 hours before surgery. After surgery, both heparin infusion and oral anticoagulants are resumed and the former is usually permanently stopped 2-4 days later when the patient's INR reaches back its therapeutic target level.

Prosthetic valve infective endocarditis occurs in 3–6% of recipients of substitute valves. Infection is generally categorized into early (less than 60 days postoperative) and late. The microbial etiology of early prosthetic valve endocarditis is dominated by staphylococcal species, *S. epidermidis* and *S. aureus*, even though prophylactic regimens used today are targeted against these microorganisms. The clinical course tends to be frequently fulminant, with rapid deterioration of the hemodynamic status due to valvular or annular destruction or persistent bacteremia. In late endocarditis, a probable source of infection can be found in 25–80% of patients, the most frequent causes being dental procedures, urological infections and interventions, and indwelling catheters. The most common organisms are *S. epidermidis, S. aureus, Streptococcus viridans*, and enterococci. In mechanical prostheses, infections involves the prosthesis-tissue junction at the sewing ring, and accompanied tissue destruction around the prosthesis; with the formation of a ring abscess, valve dehiscence, fistula, or heart block caused by conduction system damage. In addition, bioprosthetic valve infections may involve the cuspal tissue destruction with valve incompetence or obstruction. Additional complications of PVE include embolization of vegetations and congestive heart failure secondary to obstruction or regurgitation. Surgical re-intervention usually is indicated by large highly mobile vegetations, if there are cerebral thromboembolic episodes, after failure of intense medical therapy and is almost inevitable in the early type with a high mortality rate between 20 and 50%, compared to a 10-20% in the late and less progressive type.

Early endocarditis Is best avoided by strict intra operative as well as early postoperative sterilization measures. Late endocarditis can generally be avoided by prescribing a 5 days full dose broad spectrum antibiotic regimen for patients undergoing dental or other invasive procedure or instrumentation. The regimen should start 2 days before the procedure to end 2 days after and the prescribed antibiotic type is largely dependent on the commensals that are usually found in the body area being invaded for treatment.

Prosthetic valve structural deterioration can necessitate re operation or cause prosthesis-associated death. Durability considerations vary widely for mechanical valves and bioprostheses, for specific types of each, for different models of a particular prosthesis. In general, recent mechanical cardiac valves have an expected 20-30 years of structural durability, followed by homografts and autografts. Cuspal mineralization, tears and perforation are responsible for the low 7-10 years durability expected with xenografts however, the process is slowly progressive and does generate symptoms permitting re-operation; in contrast to the often catastrophic and life threatening mechanical valve failure.

Aortic dissection and Aneurysms

Relevant Anatomy: From inside to outside, the aorta is composed of the intima, media, and adventitia. The innermost thin intima is lined by endothelium and is easily traumatized. The elastic media is composed of laminated but intertwining sheets arranged in a spiral manner and, in contrast to peripheral arteries, has very little smooth muscle and collagen which allow higher distensibility, elasticity, and tensile strength. Lastly, the outermost adventitia consists of collagen and contains the vasa vasorum, which supplies blood to the outer half of the aortic wall, as well as nerve endings responsible for pain in case of aortic dissection.

The aorta has thoracic and abdominal regions. The thoracic aorta is divided into the ascending, arch, and descending segments; the abdominal aorta is divided into suprarenal and infrarenal segments. The ascending aorta is 5 cm long and is made up of a lower root and an upper tubular segments; ending just proximal to the origin of the innominate artery. The aortic root consists of the aortic valve, sinuses of Valsalva and

give openings to the left and right coronary arteries. The point of connection between the wider root and the more cylinder tubular part is called the sinotubular junction.

The arch of the aorta curves upward between the ascending and descending aorta, gives origin to the brachiocephalic (innominate, left common carotid and left subclavian) arteries and ends at the aortic isthmus. The descending aorta extends from distal to the left subclavian artery lying initially in the posterior mediastinum to the left of the course of the vertebral column, then passes in front of the vertebral column in its descent and ends behind the esophagus before passing through the diaphragm at the level of the 12th thoracic vertebra. The abdominal aorta then continues down to the level of bifurcation at the fourth lumbar vertebra and gives the splanchnic arteries.

Acute aortic dissection

Pathophysiology: The aortic wall is continuous, is exposed to high pulsatile pressure and shear stress and hence has an intrinsically high wall tension (according to Laplace law) making it particularly prone to injury. Aortic dissection -defined as separation of the layers within the aortic wall- usually starts by a tear in the intimal layer with blood entering the intima-media space (figure 8). Blood forces its way splitting the aortic wall itself, and extending both proximally and distally till finding an exit tear to join the aortic lumen again. Hence, blood is now flowing along 2 channels or lumens; the inner true (original) lumen of the aorta and, the outer (false) lumen made inside the aortic wall itself. The true lumen is lined by the original intima and contains most of the elastic media, while the false lumen is lined by only part of the elastic media and all weak the adventitia and hence, is weak and prone to progressive dilatation (aneurysm formation) and subsequent rupture.

Most classic aortic dissections begin at one of three distinct anatomic locations, including (1) approximately 2.2 cm above the aortic root, (2) the aortic arch, or (3) descending thoracic aorta distal to the left subclavian artery. As dissection can propagate both antegrade or retrograde to involve more of the aorta and compromise or occlude its branches, retrograde propagation –especially in dissection involving the ascending aorta- can lead to separation of the aortic valve or coronary ostial attachments to the aortic wall causing aortic incompetence and myocardial ischemia or infarction; respectively. The dissection usually stops at an aortic branch vessel or at the

level of an atherosclerotic plaque. The dissection can however, progress into aortic rupture and exsanguinating hemorrhage that most commonly involves the ascending aorta with the development of hemopericardium or the descending aorta with the development of left sided hemothorax.

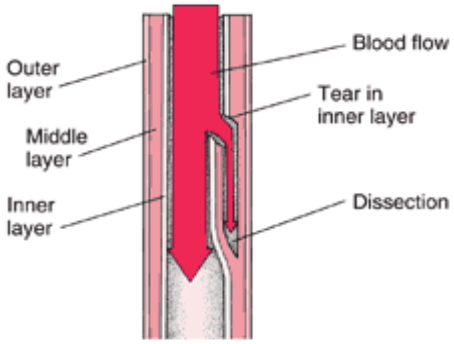

Figure 8: Aortic dissection

Etiology: The condition is more common in males than in females (2:1) and the condition commonly occurs in persons in the sixth and seventh decades of life. Naturally, patients with Marfan syndrome present earlier, usually in the third and fourth decades of life. Most (70%) of the patients are hypertensive, having their aortic wall weakened by various risk factors including; atherosclerosis (cystic medial necrosis), Marfan syndrome, aortic aneurysm, iatrogenic factors (cardiac surgery or catheterization), trauma (deceleration injury in the course of chest trauma), Ehlers-Danlos syndrome, Syphilitic aortitis, aortic arch hypoplasia, cocaine use, advanced age and pregnancy.

Clinical picture:

1) Chest pain: Classically, patients present with sudden onset of severe and tearing chest pain. As dissection advances involving more areas of the aorta and compromising their branches, the site of pain may be anterior (ascending aorta), typical myocardial (involvement of coronary arteries), affecting neck or jaw (aortic arch), Interscapular region (descending aorta), loins (renal artery involvement), abdominal (abdominal aorta and visceral branches involvement). However, pain may be mild, mimicking musculoskeletal conditions or even totally absent.

2) Neurologic deficits are present in 20% of cases. As in pain, neurological symptoms point to the areas whose arterial supply has being involved. Hence, cerebrovascular

accident (CVA), paraplegia, limb numbness, paresthesias, pain or weakness may occur due to involvement of neck, spinal and peripheral nerve arteries; respectively. Compression symptoms on cervical sympathetic ganglia and recurrent laryngeal nerve may lead to the development of Horner's syndrome and hoarseness of voice; respectively.

3) Cardiovascular manifestations include signs of aortic regurgitation (if present) and congestive heart failure, myocardial infarction (coronary involvement), SVC syndrome (compression) and limb ischemia following the occlusion of its feeding vessel. Patients usually present with the underlying essential hypertension and hence, hypotension has to be seriously evaluated. Although it can result from an excessive vagal tone but can follow cardiac tamponade or hypovolemia from the very serious aortic rupture.

4) Respiratory symptoms can include dyspnea and hemoptysis if dissection ruptures into the pleura or if tracheal or bronchial obstruction has occurred. Physical findings of a hemothorax may be found if the dissection ruptures into the pleura.

5) GIT symptoms include dysphagia, flank pain, and/or abdominal pain. Dysphagia may occur from compression of the esophagus. Flank pain may be present if the renal artery is involved. Abdominal pain may be present if the dissection involves the abdominal aorta.

Imaging diagnosis and Lab studies: Chest radiography is the initial imaging technique and may or may not reveal any abnormality: mediastinal widening, abnormal aortic contour, deviation of the trachea to the right or pleural effusion. The used imaging tool depends upon whether or not the patient is hemodynamically stable. TEE is the tool of choice for being as accurate as CT scanning MRI or Aortography in terms of sensitivity and specificity and has more advantages than other imaging techniques because it is portable (bed side), no intravenous contrast agent to inject and can be used in hemodynamically unstable patients.

The patient should be fully investigated with special emphasis on BUN and creatinine levels (renal involvement, or prerenal azotemia resulting from blood loss or associated dehydration), CK-MB and troponin I and T (coronary involvement and myocardial ischemia), hemoglobin and hematocrit values (leaking or ruptured dissection).

Medical therapy: According to the Stanford classification, aortic dissection is classified into type A and B depending on whether the ascending aorta is or not involved; respectively. Unlike DeBakey classification, this system also helps delineate treatment as type A dissections requires surgery (surgery reduces mortality to half: from 60 to 30%), whereas type B dissections are managed medically under most conditions (surgery triples mortality: from 10 to 30%).

Nevertheless, and during the acute stage (first 2 weeks of occurrence), the patient's mortality is as high as 50-90%. Hence, once diagnosed, the patient has to be admitted to the ICU with invasive blood pressure, CVP, cardiac output (Swan – Ganz) and urine monitoring. Pain is relieved by narcotics and Blood pressure is aggressively controlled with beta blockers and sodium nitroprusside infusion. TEE offers a bed-side diagnosis and type A patients are usually referred for emergency surgery. After controlling the acute episode, type B patients are usually followed up medically, through regular clinical and imaging evaluation, for strict control of hypertension and other risk factors, as well as for the development of complications; including subsequent aneurysm formation.

Surgical therapy: Surgery is indicated for all Stanford type A dissections to repair the associated severe aortic incompetence and to prevent the common rupture of the dissected ascending aorta. Surgery is however contraindicated following a major stroke, during pregnancy and in the old debilitating patient for the high morbidity associated with the use of CPB in such cases (stroke aggravation, child loss and major bodily system dysfunctions; respectively).

On the other hand, surgery in Stanford type B aortic dissections is only indicated in cases showing progressive evolution and impending rupture (progressive increase in size of aorta or accompanying hematomas, formation of a saccular aneurysm), actual rupture (progressive hypotension or shock with development of pleural effusion or cardiac tamponade), or organ (cerebral or visceral) and limb ischemia due to occlusion of a major aortic branch.

Surgical techniques: (vide-infra).

Aortic Aneurysms

Relevant Anatomy: Aneurysms are usually defined as a localized dilation of an arterial segment greater that 50% its normal diameter. The average diameter of the mid-descending thoracic aorta is 26-28 mm, compared with 20-23 mm at the level of the celiac axis and the average size for an infrarenal aorta is 2 cm. Aortic aneurysms are usually resected when their diameter reaches 5-6 cm for the possibility of rupture.

Etiology: The true etiology is probably multifactorial, and the condition occurs in individuals with multiple risk factors including; aging, smoking, hypertension, atherosclerosis, bicuspid aortic valves, and genetic disorders. Aging results in changes in collagen and elastin, which elastic fiber fragmentation and development of cystic medial necrosis. Aortic wall weakness by dissection, especially with a persistent false channel, is the second most common type. Genetics play a role as 15% of first-degree relatives of patients have an aneurysm. Patients with Marfan syndrome may develop annuloaortic ectasia of the sinuses of Valsalva, commonly associated with aortic valvular insufficiency and aneurysmal dilation of the ascending aorta. Type IV Ehlers-Danlos syndrome results in a deficiency in the production of type III collagen, and individuals with this disease may develop aneurysms in any portion of the aorta. Other causes are infection (mycotic or syphilitic), Takayasu arteritis and trauma. The abdominal aorta is by far the commonest site for aortic aneurysm formation

Clinical: Patients with thoracic aneurysms are often asymptomatic.

1) Pain is the most common presenting symptom and may be acute, implying impending rupture or dissection, or chronic, from compression or distension. As previously described, the location of pain may indicate the area of aortic involvement, but this is not always the case. In abdominal aortic aneurysm, pain is typically deep and penetrating pain mainly in the back. The pain can be severe and is usually unrelenting if the aneurysm is leaking.

2) Other symptoms: Ascending aortic aneurysm may produce aortic insufficiency with widened pulse pressure and diastolic murmur or SVC obstruction manifesting as distended neck veins. Arch aneurysms may cause hoarseness, which results from stretching of the recurrent laryngeal nerves. Descending thoracic aneurysm may compress the trachea or bronchus and cause stridor, wheezing, or cough. Compression of the esophagus results in dysphagia. Erosion into surrounding structures may result in hemoptysis, hematemesis, or gastrointestinal bleeding. Erosion into the spine may

cause back pain or instability. Spinal cord compression or thrombosis of spinal arteries may result in neurologic symptoms of paraparesis or paraplegia. Lastly all aneurysms may thrombose or embolize clot and atheromatous debris distally to visceral, renal, or lower extremities with the development of serious ischemia or gangrene.

People who have an abdominal aortic aneurysm often become aware of a pulsing sensation in their abdomen. Doctors may feel a pulsating mass in the midline of the abdomen and can sometimes even hear a bruit (turbulence of blood inside the aneurysm) with a simple stethoscope placed on the middle of the abdomen of slim patients. When the mass is becoming tender with the patient feeling severe pain in lower abdomen and back, an ongoing rupture has to be suspected.

3) The most common complications of aortic aneurysms is rupture or dissection. The appearance of ecchymoses and petechiae may be particularly challenging, because these signs probably indicate disseminated intravascular coagulation. However, the risk of significant perioperative bleeding is extremely high and large amounts of blood and blood products must be available for resuscitative transfusion.

Imaging diagnosis:

1) Chest radiograph: In the case of ascending aortic aneurysm, chest radiographs may reveal a shadow to the right of the cardiac silhouette and convexity of the right superior mediastinum. Lateral films demonstrate loss of the retrosternal air space. Plain chest radiographs may show a shadow anteriorly and slightly to the left for arch aneurysms and posteriorly and to the left for descending aorta. Aortic calcification may outline the borders of the aneurysm in the anterior, posterior, and lateral views in both the chest and abdomen.

2) Echocardiography: TEE can show and evaluate the aortic valve, ascending aorta, descending thoracic aorta, and differentiate aneurysm from dissection, but is limited in the area of the distal ascending aorta, transverse aortic arch, and upper abdominal aorta.

3) Aortography and coronary angiography is indicated in patients older than 40 years or those with a history suggestive of coronary artery disease.

4) Abdominal ultrasonography can clearly show the size of an abdominal aortic aneurysm and can be used as a follow up measure for monitoring increase in size.

5) CT scans with contrast have become the most widely used diagnostic tool. They rapidly and precisely evaluate the thoracic and abdominal aorta to determine the size, location and extent of the aneurysm and the relationship of the aneurysm to major branch vessels and surrounding structures. They can help accurately determine the presence of dissection, mural thrombus, intramural hematoma, free rupture, and contained rupture with hematoma.

6) MRI and magnetic resonance angiography have the advantage of avoiding nephrotoxic contrast and ionizing radiation compared with CT scans and supplying sometimes even superior information. However, they are more time consuming, less readily available, and more expensive than CT scans.

Medical therapy: All aneurysms must be treated with risk-factor reduction. Systemic hypertension Tobacco smoking and control of other risk factors for peripheral arterial obstructive disease may be beneficial. Aneurysms that are less than 5 centimeters wide rarely rupture. Imaging procedures are performed every 3 to 6 months to estimate the rate of enlargement and determine when surgery will be necessary.

Surgical therapy: excision and graft replacement is based on the size or the growth rate and symptoms. Because the risk of rupture is proportional to the diameter of the aneurysm, patients with aneurysms reaching 6 cm (widest diameter) are candidates for elective surgical repair. Patients with Marfan syndrome or familial aneurysms have a tendency of earlier rupture and should undergo earlier repair when the aneurysm diameter reaches 5 cm.

Rapid expansion is also a surgical indication and a growth rate of 1 cm/y or faster is an indication for elective surgical repair. Symptomatic patients should undergo aneurysm resection regardless of size and acutely symptomatic patients (e.g. rupture) require emergent operation.

The hospital mortality of chronic aneurysms largely varies on location of aneurysm as well as the surgeon’s experience; being the lowest (2-5%) in abdominal aortic aneurysm, 5-15% in ascending or descending aortic aneurysms, 25% in arch and as high as 25- 50% in thoracoabdominal aneurysms. Ruptured aortic aneurysm is a

surgical emergency that is associated with not less than 50% mortality rate, in best hands.

Techniques of aortic surgery:

Besides a perfect anesthetic technique, common rules of aneurysm surgery include measures for blood conservation, for the associated pre, intra as well as postoperative considerable blood loss and transfusion. These include the use of a cell saver mechanism which permits washing of the red blood cells of shed blood before re transfusing them back to the patient. The use of Aprotinin (trasylol) in a dose of 1.5 million units (given in 3 divided doses during surgery) was found to decrease postoperative bleeding considerably. The reversal of heparin with protamine sulfate is always necessary after thoracic aortic surgery, even if CPB was used and unless indicated, profound hypothermia is better avoided –or at least shortened- during CPB, for the associated increase in intra as well as postoperative bleeding. Beside the use of fresh frozen plasma, it became evident to transfuse large amounts of platelets that are largely consumed during CPB. Locally, a less then blood-tight suture lines is not permissible and the use of collagen impregnated grafts which pores become fluid seal, Teflon to buttress suture lines and biological glue to close dissected wall and to consolidate tissues made friable by dissection; all are indispensable tools.

1) **The approach of ascending aorta and arch** is through median sternotomy. CPB is instituted and the ascending aorta and/or arch are resected and replaced with a synthetic collagen impregnated (more hemostatic) Dacron patch. In case of dissection, and according to the extent of the lesion, the involved aortic valve is re suspended (or replaced by prosthesis), coronary openings and brachiocephalic vessels are re sutured to the aortic wall and reinforced by biological glue and Teflon pledgets. Moreover, and in order to consolidate the friable aortic tissues, the separated intima-media space is glued by biological glue and the suture lines are reinforced by Teflon pledgets.

As periods of total circulatory arrest are usually needed during part of the process, measures are used for brain protection; the commonest of which includes: cooling of the patient (and consequently the brain) by the CPB system down to about 18 C for minimizing cerebral metabolic needs. Then arresting the circulation; with the cerebral tissues being infused retrogradely via a cannula placed in the superior vena cava. Such

maneuver usually permits a safe one hour period of total circulatory arrest without the need of other measures of protection for other body organ.

2) **The descending thoracic aorta** is approached through a posterolateral thoracotomy without the need of CPB. The thoracic aorta is clamped both proximally and distally of the lesion, and thus creating hypertension proximally (heart is still beating and all cardiac output is directed solely to head and arms) and severe hypotension and ischemia both in clamped area (spinal cord blood supply through branches of clamped thoracic aorta) as well as distally (no blood is perfused to abdominal organ and lower limbs).

In this setting, graft replacement can either be carried while controlling proximal hypertension with beta-blockers and sodium nitroprusside infusion and reducing spinal cord ischemia with various measures including; lowering the patient's temperature to 32 C (cooling the operating room and using cooling blankets), spinal fluid drainage (to decrease harmful increased pressure induced by aortic clamping) infusing ice cold ringer's lactate locally in the aneurysm and before opening it, etc…Besides heparinizing the patient for prevention of local as well as distal thrombosis and/or embolism, those measures provide a safe and sufficient one hour period.

The other way to do it is to supply the viscera distal to the anastomosis with blood by placing a cannula in the left pulmonary artery that will drain venous blood into the CPB machine. As usual blood is oxygenated and pushed back into the patent's femoral artery to retrogradely perfuse the lower body rendered ischemic by clamping the descending thoracic aorta. As this CPB has the aim to only perfuse the lower body, it is called partial bypass. Although looking safer than the previous method, yet more coagulation disturbances are induced by the use of CPB in patients who are already at a high risk of bleeding. Nevertheless, the descending thoracic aorta is replaced by a collagen impregnated Dacron graft, in which a side hole is made to re implanted the intercostal arteries. Same previously mentioned precautions in 1) are taken in case of dissection.

3) Abdominal aortic aneurysm: through a middle line abdominal incision, the infra renal abdominal aorta is carefully dissected, the aneurysmal portion is isolated between vascular clamps, incised longitudinally and the bleeding posterior branches are secured with stitches. A suitable sized impregnated Dacron graft is then anastomosed to the

upper and lower (normal) ends of the abdominal aorta and the remaining aneurysmal tissue is then wrapped around it for isolation and prevention of any gut adherence with possible erosion in the future. A common distal extension of the aneurysmal disease to the iliac or femoral arteries necessitates the use of a bifurcated graft. The single proximal end is sutured (end to end) to the lumen of abdominal aorta above the aneurysm and both smaller distal ends are sutured either to the iliac arteries or, through bilateral groin incisions to the femoral arteries.

As such, surgery is simple and rewarding however, the procedure becomes more complicated when the disease extends upwards to the renal or celiac portions of the aorta. Isolation of those parts, re implantation (or inclusion) of celiac and renal arteries to the graft are more demanding, and problems related to renal ischemia may appear.

4) The Thoracoabdominal aorta is accessed via a thoracoabdominal incision that involves circumferential incision of the diaphragm. The viscera are approached retroperitoneally and pushed forwards and the thoracoabdominal aorta is totally replaced followed by re implantation of the thoracic intercostal, renal and visceral arteries to side holes made inside the graft. As for the thoracic type, the procedure can either be approached with or partial bypass to protect spinal cord and viscera rendered ischemic by aortic clamping; however such surgery is more demanding for the length of the procedure and the extent of involved organs.

5) Minimal invasive aortic aneurysm surgery: classical procedures take hours with extended hospital stay and the much less invasive approach “stent grafting” is indicated in selected patients. Under regional or epidural anesthesia, a small incision is made in the groin. With fluoroscopic guidance, a catheter containing the stent-graft (which resembles a meshed, collapsible straw) is then slipped over a guide wire and positioned inside the aneurysm. Then the stent-graft is opened, forming a stable channel for blood flow. The procedure takes 2-5 hours; the hospital stay is usually 2-5 days and the mortality risk is about 2-5%.

Surgery of congenital heart diseases

According to the arterial oxygen saturation, congenital heart diseases are classically classified into an acyanotic (normal saturation) and a cyanotic (reduced

saturation) group. Acyanotic heart diseases include: Ventricular septal defect (VSD), atrial septal defects (ASD), patent ductus arteriosus (PDA), pulmonary stenosis, coarctation of the aorta, endocardial cushion defect, etc… Cyanotic heart diseases include: tetralogy of Fallot, transposition of the great arteries, tricuspid atresia, total anomalous pulmonary venous drainage, truncus arteriosus, hypoplastic left or right heart, Ebstein's anaomaly etc…These abnormalities may occur as single defects or in various combinations. VSD is the most commonly diagnosed congenital heart defect (about one-third of all cases) and it is seen almost three times as often as ASD and PDA, which are the next most common. The majority of congenital heart diseases occur as an isolated defect and are not associated with other diseases. However, they can also be a part of various genetic and chromosomal syndromes, such as Down syndrome, trisomy 13, Turner's syndrome, Marfan syndrome, Noonan syndrome.

Surgery of congenital heart diseases may be divided into procedures permitting complete repair and those intending to palliate the congenital anomaly. Palliative procedures are sometimes definitive for the complexity of the disease as in the case of a rudimentary right ventricle. Most palliative procedures however, are temporary and are either carried out in young children first presenting in a bad general condition or heart failure that largely compromise the results of a definitive repair or in cyanosed infants in whom palliation aims to allow somatic growth and hence, improving the chances of success of a subsequent total repair. In fact, surgery in the young infant is a challenge to all: surgeon, anesthesiologist, CPB operator and intensivist.

Complete repair is feasible in many extra cardiac as well as intra cardiac congenital anomalies. Examples of the former are: division of a patent ductus arteriosus and repair of aortic coarctation and are almost always carried out without CPB; with minimal hospital morbidity and mortality. Intra cardiac anomalies however, usually necessitate CPB and the result of repair is largely depending upon the complexity of the lesion, the child age and the experience of the operating team.

Repair of extra cardiac congenital anomalies: The commonest anomalies are patent ductus arteriosus (PDA) and coarctation of the aorta; both of which are approached through a left thoracotomy. PDA is usually divided between clamps or ligated; while coarctation is relieved through different procedures; including resection followed by re anastomosis of cut aortic ends, dilatation of the stenosed (coarcted) segment by the use of a subclavian artery flap or synthetic Dacron patch and lastly, in some adult cases, the stenosed segment can be simply bypassed by a suitable sized ($\geq$20 mm)

Gortex tube which proximal end is implanted on the subclavian artery (feeding artery) and the distal end implanted on the descending thoracic aorta.

Surgery of aortic coarctation involves clamping of the descending thoracic aorta and sometimes the subclavian artery and hence, interferes with the blood supply of the spinal cord during the clamping period. Fortunately, postoperative paraplegia is extremely rare; being only recorded in few redo or adult cases and hence, measures used for cord protection are rarely undertaken (see surgery of thoracic and thoracoabdominal aneurysms for details).

Palliative procedures for intra cardiac congenital anomalies as a step towards final repair are usually indicated in patients with cyanotic heart diseases to relieve cyanosis and hence, to allow the patient to grow so as to fulfill the best criteria for a successful final repair. The commonest example is the Blalock-Tausig shunt used for palliation of infants and young children presenting with Fallot's tetralogy. Classically, the procedure involves the implantation of the distal cut end of the left subclavian artery into the left pulmonary artery, via a left thoracotomy incision. As the procedure is carried out in the young, left arm ischemia is extremely rare for the rich anastomosis around the scapula. The increased pulmonary blood flow through the subclavian artery alleviates cyanosis, permits the child to grow and is said to enlarge the stenosed pulmonary outflow; all of which improves the chances for a successful total repair in the future.

Actually, a modified procedure (modified Blalock Tausig shunt) involves the use of 5 or 6 mm Gortex tube that is implanted proximally on the subclavian artery and distally on the pulmonary artery (a bypass to the stenosed pulmonary tract) is the actual common practice for being technically easy, used on either sides of the circulation (left or right), convenient for older children for not interfering with arm circulation and lastly, for being easier to close during a future total repair surgery.

Complete repair of intra cardiac congenital anomaly is usually carried under CPB. The commonest example is the total repair of Fallot's tetralogy that accounts for 15% of all cyanotic heart diseases. The anomaly compromises (i) a mal alignment VSD (ii) with the aorta straddling the defect and over riding both ventricles (iii) right ventricular hypertrophy and (iv) pulmonary tract stenosis. The aim of the repair is to close the intra cardiac left to right shunt (VSD) responsible for the cyanosis; which is usually done by a synthetic Dacron patch. At this point however, the right ventricle blood –on its way to the pulmonary circulation- is faced by 2 obstructions made by a hypertrophied right ventricular outflow and a stenosed pulmonary tract; both of which have to be relieved

with or without the use of a widening pericardial patch. Improperly done, right ventricular failure supervenes in response to those obstructions. The results of surgery are dramatic and gratifying with disappearance of cyanosis, a significant increase of patient's tolerance to exercise and a long-term follow-up demonstrating continued good health.

Cardiopulmonary resuscitation (CPR)

In unconscious or collapsed persons, the state of ventilation and circulation must be determined immediately. Speed, efficiency, and proper application of CPR directly relate to successful neurologic outcome as tissue anoxia for more than 4 to 6 min can result in irreversible brain damage or death; yet prognosis varies widely depending on age, cause of arrest, and clinical circumstances. CPR must be continued until the cardiopulmonary system is stabilized or the patient is pronounced dead Guidelines established by the American Heart Association divide CPR into a primary and secondary survey that are performed in a hospital facility or equipped ambulance vehicles (table 1).

Table 1: THE ABCDs OF CARDIOPULMONARY RESUSCITATION

Primary Survey (Basic Life Support)	Secondary Survey (Advanced Cardiac Life Support)
A Airway opened Establish airway patency using head tilt–chin lift, head tilt–neck lift, or mandibular jaw thrust If available, use an artificial airway in the unconscious patient	**Airway opened** Maintain airway patency Progress to endotracheal intubation if experienced personnel are available
B Breathing restored Note chest movement If no spontaneous chest movement, initiate mouth-to-mouth rescue breathing Reassess for chest movement	**Breathing restored** Note chest movement If endotracheal intubation is accomplished, assess patient for current position of endotracheal tube
C Circulation restored Establish pulselessness If pulseless and appropriate equipment is available, rapidly assess for VF or pulseless VT If VF or pulseless VT is present, defibrillate If pulseless and appropriate equipment is not available, begin chest compressions	**Circulation restored** Establish IV access Accurately assess vital signs Provide appropriate drug therapy based on rhythm and vital signs If pulseless and appropriate equipment is available, rapidly assess for VF or pulseless VT If VF or pulseless VT is present, defibrillate
D Defibrillate	**Differential diagnosis** Consider underlying cause of cardiac arrest and implications with respect to drug therapy

VF = ventricular fibrillation; VT = ventricular tachycardia.

PRIMARY SURVEY or BASIC LIFE SUPPORT (BLS)

After establishing unresponsiveness of the victim (tap, shake, or shout), the rescuer calls for assistance, notes the exact time of arrest (if known), and positions the victim horizontally on a hard surface. Then BLS is rapidly carried out following the mnemonic ABC (Table 1).

A) Opening the airway is the first priority and is sometimes all that is needed to restore spontaneous breathing (B) and circulation (C). Airway obstruction resulting from relaxed tongue and neck muscles in an unconscious person is accentuated with neck flexion and a (head tilt - chin lift) is performed by tilting the head back and placing a finger of the second hand under the rim of the mandible, lifting the chin forward (vertically upwards) until the teeth are brought almost together. If unsuccessful, the (head tilt - neck lift) should be used by placing one hand on the victim's forehead, lifting the neck straight up while tilting the head back. A mandibular jaw thrust with only chin-lift (without neck or head tilt) is indicated in trauma patients with suspected cervical spine injuries. In chest trauma patients however, stabilization in the field by trained medical personnel and immediate transport to a specialized facility are indicated.

B) Breathing is restored using mouth-to-mouth resuscitation by placing the heel of one hand against the victim's forehead to keep the head tilted backward, using the thumb and index finger to gently pinch the nostrils shut to prevent escape of air. The rescuer opens his or her own mouth widely, deeply inhales, makes a tight seal with the mouth over the victim's mouth, and blows two full breaths (1 to 1.5 sec each), thereby beginning ventilation yet avoiding trapping air in the stomach. The adequacy of these ventilatory efforts is assessed by seeing the victim's chest rise and fall and by hearing and feeling passive exhalation. Adequate time (1 to 2 sec per ventilation) should be provided to allow for exhalation.

Mouth-to-nose resuscitation is indicated when a tight seal around the victim's mouth is impossible and combined mouth-and-nose resuscitation is used for infants and small children when a tight mouth seal cannot be maintained. If the airway is assumed to be still obstructed, the victim is rolled into the supine position, and the Heimlich maneuver (manual thrusts to the upper abdomen or, in the case of pregnant or extremely obese patients, chest thrusts performed as for cardiac compression) should be given. Sitting astride the unconscious victim (above the knees) and placing the heel of a hand below the xiphoid process; the other hand is placed on top of the first and a firm upward thrust is delivered. Six to 10 thrusts may be necessary to dislodge a foreign body. If obstruction persists, cricothyrotomy or tracheotomy must be performed.

C) Circulation Restored: While tilting the victim's head backward to open the airway, the rescuer should use a free hand to gently palpate for the carotid pulse; which absence alarms for the need of an immediate external cardiac massage alongside rescue breathing. With the middle finger in the xiphisternal junction, the rescuer places the index finger on the lower end of the sternum and the heel of the other hand on the sternum just above the index finger of the palpating hand. The heel of the palpating hand is then placed on top of the hand on the sternum *-not the xiphoid process-* to begin compressions. The rescuer should be positioned directly over the victim and, keeping the arm straight, should exert sufficient force directly downward over the sternum (to avoid rib fractures), depressing the sternum 4 to 5 cm in the adult. Compression time should equal release time and the rescuer's hands should remain on the sternum during the release phase. This cycle should be repeated smoothly; jerky, bouncing, or irregular compressions increase the chance of injuries.

In children between 1 and 8 yrs, the heel of one hand is used to perform external cardiac compression over the lower sternum; the depth should be 2.5 to 4 cm at a rate of 80 to 100/min. An infant's heart is higher in the chest, and the chest wall is more pliable. For compression, the tips of the index and middle fingers are used over the midsternum to a depth of about 1.3 to 2.5 cm at a rate of 100/min.

The effectiveness of CPR should be monitored periodically during resuscitation efforts through carotid palpation and restored pupillary responsiveness is a sign of adequate brain circulation and oxygenation. Complications of cardiac massage include laceration of the liver (the most serious) that is usually caused by pressing too low on the sternum, delayed rupture of the spleen, rupture of the stomach, fracture rib, heart or lung compression injury and aspiration pneumonia following regurgitation and aspiration of gastric contents. With the exception of the freshly operated upon cardiac patients, failure of closed message is not an indication for the technically demanding open cardiac massage.

D) Defibrillation: A forceful precordial thump can convert ventricular fibrillation (VF) or ventricular tachycardia (VT) into functional cardiac rhythm or vice versa; being only advised when a defibrillator is not available. In fact, prompt DC cardioversion appears to be more effective in resuscitation than do antiarrhythmic drugs; however, the success of defibrillation is time-dependent, with a 2 to 10% decline in success rate per minute of cardiac arrest

Defibrillating paddles (with conducting paste or moist saline pads beneath them) are placed over the 2nd intercostal space along the right sternal border and over the 5th or 6th intercostal space at the apex of the heart. If an immediate countershock of 200 joules is unsuccessful, a second countershock of 200 to 300 joules is given followed by a third countershock of 360 joules if VF persists. These 3 countershocks should be delivered consecutively, without interruption for CPR or drug therapy. The defibrillating paddles should be immediately recharged after each countershock without removal from the chest wall if conventional paddles are used. If unsuccessful, BLS should be resumed, and drug therapy is based on the secondary survey (vide-infra).

SECONDARY SURVEY

The same ABCs are followed however; the presence of facilities (in hospital or ambulance) provides equipment for better airway control (e.g. endotracheal or tracheostomy tubes) and disease – tailored oxygen supply (e.g. special mask). Better diagnosis (ECG monitoring) and treatment of arrhythmia (drugs, DC shock, etc...) as well as methods to ensure hemodynamic stability (e.g. IV access lines to infuse blood or fluids, blood gas analysis, etc…). Most importantly, ACLS has to provide the differential diagnosis of the cause of arrest as causes such as electrolyte abnormalities (hypokalemia or hyperkalaemia), acid-base disorders (metabolic acidosis), hypovolemia, massive pulmonary embolism, or pneumothorax that will dictate the specific therapy to be used. Table 1 shows the sequence of ACLS however, and as word of caution; the implementation of ACLS should not interrupt BLS for more than 15 to 30 sec. While the most important ECG monitoring cannot interrupt other procedures, an IV access line can sometimes be a time consuming procedure that requires interrupting BLS. In this setting, remember that the most commonly used drugs as lidocaine, atropine, and epinephrine may be given via the endotracheal tube at 2 to 2.5 times the IV dose. Nevertheless, a single, rapid, large bored and short IV line placed in an antecubital vein permits the delivery of large volumes of blood or fluid when needed and is preferred over the time consuming several short and/or a single long venous line. A subclavian or internal jugular central line however, can be placed by experienced personnel, as patients subjected to CPR should never be used for training purposes.

1- Drug Therapy

a) VF and VT: Epinephrine (1 mg in 10 ml q 3-5 min) is the first-line drug when initial defibrillation fails and rhythm is reassessed 30-60 sec later. Unsuccessful, up to 3 consecutive cardioversions can be given before further drug therapy. Unsuccessful, this sequence is repeated before the use of any additional drugs. Unsuccessful, the following drugs can be given in consequence; each followed by cardioversion after 30-60 seconds. Lidocaine 1.0 to 1.5 mg/kg is given rapidly IV and can be repeated after 3 min; its success however, requires constant infusion to maintain therapeutic blood levels. Bretylium tosylate and procainamide are not available in many hospitals.

Its worth noting that sodium bicarbonate is no longer recommended as initial, automatic therapy for cardiac arrest because it may induce paradoxical acidosis of the brain and heart, hyperosmolarity, or hypernatremia or alkalemia and may inhibit the

release of O_2 by the blood; and is only given when cardiac arrest is caused by preexisting bicarbonate-responsive acidosis as in hyperkalaemia or tricyclic overdose. Other specific drugs include: Phenytoin given in digitalis toxicity that is refractory to other drugs, magnesium sulfate in patients with known or suspected Mg deficiency as alcoholism and calcium chloride given in cases with hyperkalemia, hypocalcemia, or Ca blocker toxicity.

b) Asystole: A flat line on the ECG is most commonly due to operator error (false asystole) from leads that are loose or not connected to the patient or monitoring equipment, lack of power, or low signal gain and changing to another lead or reorientation of the defibrillation paddles is appropriate. Nevertheless, and again, the first choice is epinephrine 0.5 to 1.0 mg given rapidly IV q 5 min, Besides its β-adrenergic receptor properties, the α- effects of epinephrine augment peripheral and coronary diastolic pressure, thereby increasing perfusion to subendocardial regions increasing the chance to generate electrical activity. Atropine sulfate 0.5 to 1.0 mg q 5 min can also be given if asystole persists. Atropine is also useful for bradyarrhythmias with myocardial ischemia or high-degree atrioventricular nodal block.

If both drugs fail to restore regular ECG complexes, temporary transcutaneous pacing should be instituted immediately. A temporary transvenous or transthoracic pacing electrodes placed subcostally can be substituted, if transcutaneous pacing is unavailable or unsuccessful. Unfortunately, pacing is less successful the longer the cardiac arrest. Moreover, routine defibrillation of asystole is discouraged, because it can result in profound parasympathetic discharge.

c) Pulseless electrical activity is circulatory collapse that occurs despite satisfactory electrical complexes on the ECG. This may be caused by pump failure from extensive myocardial dysfunction, profound loss of peripheral vasomotor tone, massive volume loss, cardiac tamponade, intracardiac tumor or thrombus impaction, tension pneumothorax, massive pulmonary embolus as well as drug overdose with tricyclic antidepressants, digitalis, β-blockers, or Ca blockers. BLS should be combined with volume infusion, epinephrine (0.5 to 1.0 mg IV), atropine for bradycardia and other ACLS measures. Other specific measures include pericardiocentesis for cardiac tamponade, immediate needle or chest tube insertion for tension pneumothorax, etc…

d) In circulatory shock, if left ventricular failure is not evident, initial treatment is with cautious IV volume infusions. For severe arterial hypotension unresponsive to volume replacement, the following drugs are useful by continuous infusion with titration as needed to restore blood pressure: the inotrope dopamine beginning at 3 to 5 µg/kg/min; the inotrope and vasoconstrictor epinephrine given at 2 to 10 µg/min; the peripheral vasoconstrictors: norepinephrine given at 2 to 15 µg/min; or phenylephrine given at 0.1 to 1.5 µg/kg/min.

2- Mechanical Resuscitation Devices should be available in clinics and hospital areas where the need for CPR is predictable and the risk of HIV, hepatitis, or other infection is high (e.g. ICU, emergency room, operating room).

a) Airway support: Bag-valve-mask devices incorporate a self-inflating bag and a non rebreathing valve mechanism (Ambu bag) and are best used with artificial airways; only when the patient is unconscious. This is because airway obstruction and subsequent hypoxemia, vomiting, and aspiration may occur if an airway is forced into a conscious or stuporous patient's mouth.

Cuffed endotracheal tubes offer a secure airway, prevent aspiration, initiate mechanical ventilation, and permit suction of the lower respiratory tract. They are indicated in comatose patients and those in whom artificial ventilation is required. Before attempting tracheal intubation, patient is ventilated manually with oxygen and air passages are cleared out with suction. Orotracheal intubation is faster than nasotracheal intubation in emergencies, however, facing a deformity or muscle spasm blind nasotracheal intubation may be attempted. Unsuccessful, special techniques to gain airway control (e.g. transtracheal catheter ventilation, cricothyrotomy) are required.

On the other hand, nasal catheters and several types of face masks for the patient who is breathing spontaneously are available in child and adult sizes. They must fit well and be capable of delivering an O_2 concentration of 50% with an O_2 flow rate of 10 L/min. In patients with CO_2 retention and chronic lung disease, Venturi masks can provide O_2 concentrations of 24, 28, 31, 35, 40, and 50%. On the other hand, a nonrebreathing mask, similar to a simple mask but with an O_2 reservoir bag and one-way exhalation valves, can deliver tracheal O_2 concentrations of up to 90%.

b) Artificial circulatory support: Intra-aortic balloon counterpulsation pumps **(IABP)** can assist low-output circulatory states due to significant refractory left ventricular pump failure. A catheter is usually introduced via the femoral artery, percutaneously or by arteriotomy, pushed retrograde into the thoracic aorta till just distal to the left subclavian artery; for not to be occluded when the balloon inflates. In fact, the catheter carries a balloon that is inflated with helium to prevent gas embolism in the case where the balloon ruptures and the gas escapes. Through a computerized device, the balloon inflates inside the descending thoracic aorta during diastole and empties during systole; thus creating a pulsatile pressure that counteracts (not contradicts) the physiologic myocardial pulsatile flow: the generated pulsation (during balloon inflation in diastole) augments coronary artery perfusion (which fill in diastole too); and its deflation decreases afterload against which the heart is beating during systole. Hence, IABP increases coronary perfusion and decreases afterload; optimizing the conditions of myocardial contractility. Candidates are patients with cardiogenic shock after resuscitation; having lesions that are potentially correctable by surgical intervention (e.g. acute MI with acute mitral insufficiency or ventricular septal defect, severe aortic insufficiency due to acute vascular lesions). IABP is a basic equipment of any CCU or cardiac surgery theater.

3- Post resuscitative Care focuses on correction of factors that may jeopardize cardiovascular function and on standard measures to ensure optimal brain oxygenation and circulation. Blood volume should be restored, and mean arterial pressure should be normalized or raised slightly above normal. Hematocrit, serum glucose, and electrolytes must be monitored, and fever should be reduced to decrease metabolic demands. Arterial Pa_{O2} should be kept at normal values (80 to 100 mm Hg). Every effort should be made to diagnose the cause of arrest and the hemodynamically stable patient is then safely transferred (monitored, ventilated if needed, on continuous drug therapy, etc...) to the special hospital unit; accordingly.